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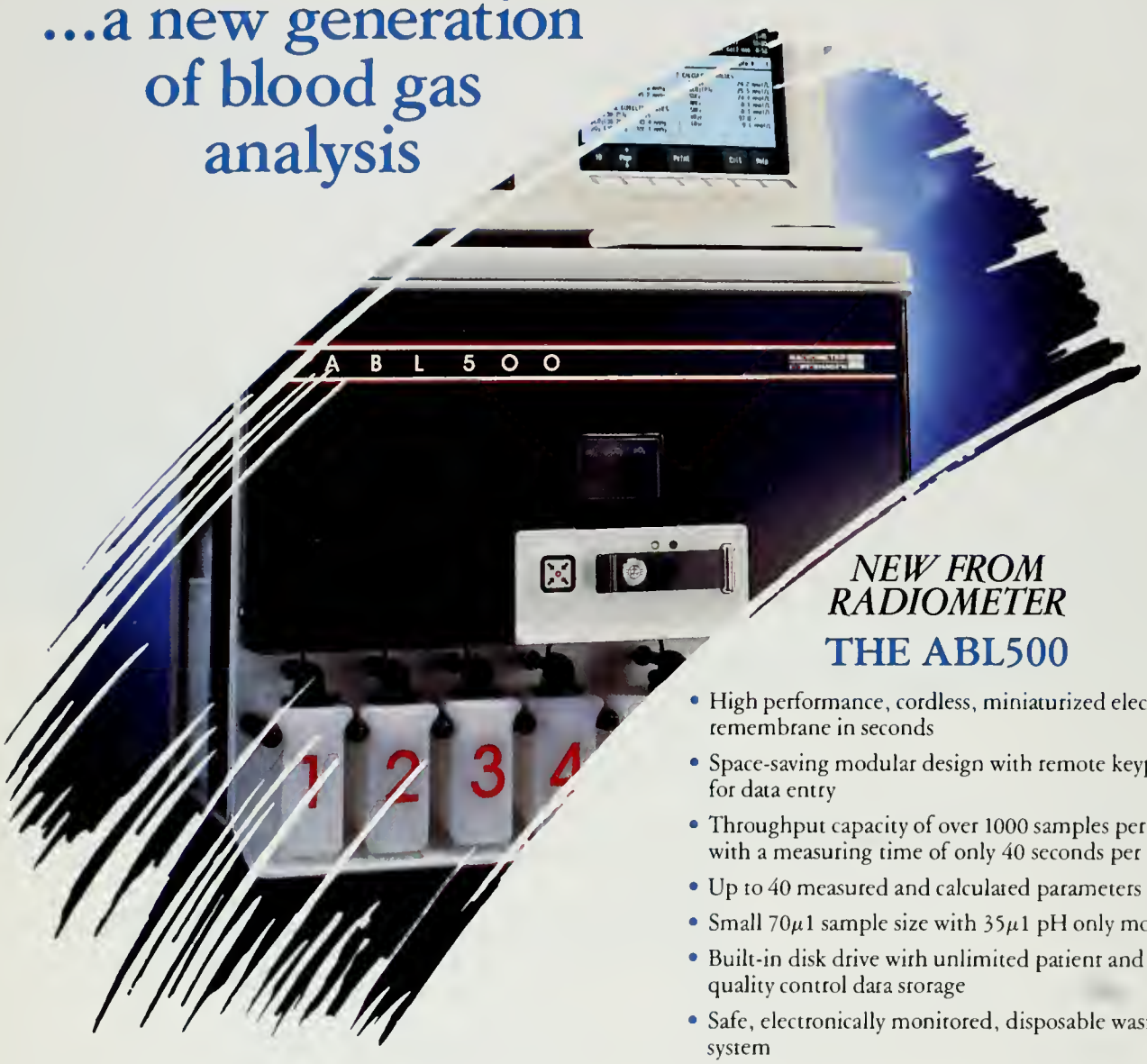

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Carcinogenesis Long-term studies in mice and rats to evaluate the oral carcinogenic potential of metaproterenol sulfate have not been completed. Studies of metaproterenol sulfate have not been conducted to determine mutagenic potential or effect on fertility.
Pregnancy *Teratogenic Effects. Pregnancy Category C.* Alupent has been shown to be teratogenic and embryocidal in rabbits when given orally in doses 620 times the human inhalation dose and 62 times the human oral dose. There are no adequate and well-controlled studies in pregnant women. Alupent should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Oral reproduction studies in mice, rats and rabbits showed no teratogenic or embryocidal effects at 50 mg/kg, corresponding to 310 times the human inhalation dose and 31 times the human oral dose. Teratogenic effects in the rabbit included skeletal abnormalities and hydrocephalus with bone separation.
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Pediatric Use Consult package insert for age limit.

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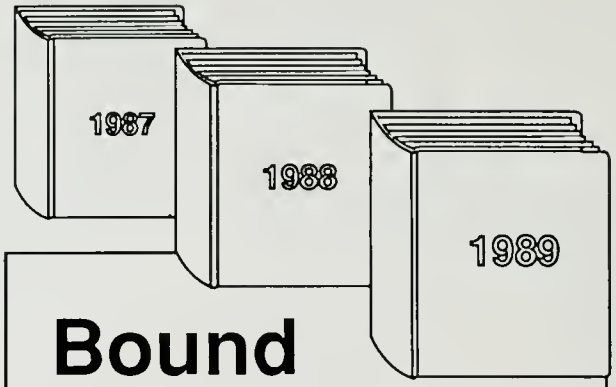

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Prolonged Relaxation Rate of Inspiratory Muscles in Patients with Sleep Apnea—GA Griggs, LJ Findley, PM Suratt, SA Esau, SC Wilhoit, DF Rochester. Am Rev Respir Dis 1989;140:706.

To evaluate whether inspiratory muscle function is impaired in patients with sleep apnea, we measured inspiratory muscle strength and relaxation rate before and after sleep in 13 patients. The sleep apnea group was composed of 8 patients with severe obstructive sleep apnea, and the non-apnea group was composed of 5 patients without significant sleep apnea. We chose the time constant of relaxation (TauR) as an index of impaired inspiratory muscle contractility, and in subsets of each group, we measured the inspiratory pressure-time index as an indicator of a fatiguing breathing pattern. In patients with sleep apnea, presleep TauR was 79 $\pm$ 22 ms (SD), longer than that of normal subjects (normal, 59 $\pm$ 7 ms) ($p < 0.05$). TauR increased by 21 $\pm$ 16 ms during sleep ($p < 0.01$). In patients without apnea, presleep TauR was 67 $\pm$ 7 ms and it did not change after sleep. Maximal inspiratory and expiratory pressures were unchanged after sleep. We conclude that patients with sleep apnea do not develop overt inspiratory muscle failure but do have impaired contractility. We speculate that hypoxemia as well as increased work load was responsible.

Effects of Respiratory Drive on Upper Airways in Sleep Apnea Patients and Normal Subjects—F Series, Y Cormier, M Desmeules, J LaForge. J Appl Physiol 1989;67: 973.

We compared the changes in nasal and pharyngeal resistance induced by modifications in the central respiratory drive in 8 patients with sleep apnea syndrome (SAS) with the results of 10 normal men. Upper-airway pressures were measured with two low-bias flow catheters; one was placed at the tip of the epiglottis and the other above the uvula. Nasal and pharyngeal resistances were calculated at isoflow. During CO_2 rebreathing and during the 2 min after maximal voluntary hyperventilation, we continuously recorded upper-airway pressures, airflow, end-tidal CO_2 and the mean inspiratory flow (V_T/T_I); inspiratory pressure generated at 0.1 s after the onset of inspiration ($P_{0.1}$) was measured every 15-20 s. In both groups upper-airway resistance decreased as $P_{0.1}$ increased during CO_2 rebreathing. When $P_{0.1}$ increased by 500%, pharyngeal resistance decreased to 17.8 $\pm$ 3.1% of baseline values in SAS patients and to 34.9 $\pm$ 3.4% in normal subjects (mean $\pm$ SE). During the posthyperventilation period the V_T/T_I was accompanied by an increase in upper-airway resistance. When the V_T/T_I decreased by 30% of its baseline level, pharyngeal resistance increased to 319.1 $\pm$ 50.9% in SAS and 138.5 $\pm$ 4.7% in normal subjects ($p < 0.05$). We conclude that (1) in SAS patients, as in normal subjects, the activation of upper-airway dilators is reflected by indexes that quantify the central inspiratory drive and (2) the pharyngeal patency is more sensitive to the decrease of the central respiratory drive in SAS patients than in normal subjects.

Smoking as a Risk Factor for Cerebral Ischaemia—GA Donnan, MA Adena, HM O'Malley, JJ McNeil, AE Doyle, GC Neill. Lancet 1989;2:643.

To assess whether a rigorous clinical classification, based on computerized tomography, of patients with cerebral ischaemia would identify subgroups at higher or lower risk with respect to cigarette smoking habits, a case-control study was carried out on 422 cases of first-episode cerebral ischaemia matched for age and sex with 422 community-based neighbourhood controls. Patients with ischaemic stroke due to extracranial or intracranial vascular disease were at higher risk from smoking than has previously been reported for stroke (relative risk 5.7, 95% confidence interval 2.8, 12.0) whereas those with stroke due to cardiac emboli had no excess risk associated with smoking (relative risk 0.4 [0.1, 1.8]). After cessation of smoking, the relative risk declined gradually over 10 years, at the end of which time a significant risk was still evident. This finding may imply that the risk incurred by smoking is due mainly to atheroma formation, rather than transient haematological effects. Exposure to smoking by a spouse was an independent risk factor for the whole group of cerebral ischaemia patients (relative risk 1.7 [1.1, 2.6]), but this was not so for smoking by either parent (relative risk 1.2 [0.8, 1.8]). These findings suggest that smoking is a more potent risk factor for the most common form of ischaemic stroke than has previously been appreciated. The persistent nature of the risk even after cessation of smoking and the possible risk associated with passive exposure strengthens public health arguments against smoking.

Duration of Action of Inhaled Terbutaline at Two Different Doses and of Albuterol in Protecting against Bronchoconstriction Induced by Hyperventilation of Dry Cold Air in Asthmatic Subjects—J-L Malo, H Ghezzo, C Trudeau, A Cartier, J Morris. Am Rev Respir Dis 1989;140:817.

Although several studies have examined the duration of the bronchodilator

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effect of several inhaled β_2-adrenergic agents, the duration of the blocking effect on bronchial hyperresponsiveness, another key feature of asthma, has seldom been studied. We investigated this problem in 8 adult asthmatic subjects who underwent hyperventilation tests with dry cold air on 44 different days. On the first day, five hyperventilation tests with assessment of the level of ventilation causing a 20% fall in FEV_1 (PD_{20}) were obtained to evaluate the within-day variability of the test. On the three other visits, after a baseline hyperventilation test, albuterol 200 μg, terbutaline 500 μg, and terbutaline 1,500 μg were administered in a double-blind, randomized way. Hyperventilation tests were carried out 1, 2, 4, and 6 h later. The blocking effect on the treatment days, as assessed by the differences in PD_{20} for each test compared with baseline PD_{20} for that day, was corrected for the within-day variability of the control day. There was a significant bronchodilator effect 1 h after administering the drug; it was equivalent for albuterol 200 μg (25.6 $\pm$ 14.7%) and terbutaline 1,500 μg (21.7 $\pm$ 13.5%) and significantly less for terbutaline 500 μg (14.1 $\pm$ 10.0%). Complete or partial blockade on bronchial responsiveness was obtained in the majority (6-7 of 8) of the subjects 1 h after inhaling the bronchodilator, with progressive reduction in the effect later on. Four subjects still showed a blocking effect 6 h after terbutaline 1,500 μg was administered, one subject after terbutaline 500 μg, and no subjects after albuterol (chi square = 6.6, p = 0.04). We conclude that the blocking effect of inhaled β_2-adrenergic agents on bronchial hyperresponsiveness of asthmatic subjects is time dependent and can last as long as 6 h in some subjects.

International Comparisons in COPD Mortality—TJ Thom. Am Rev Respir Dis 1989;140:S27.

In 31 developed countries, chronic obstructive pulmonary disease (COPD) and allied conditions comprise a major cause of death, but they cause less than 10% of all deaths, even in older persons. COPD mortality is highest in the Eastern European countries and Ireland, Scotland, and England/Wales, and is lowest in southern Europe, Japan, and Israel. That these differences are large and real is a likelihood but not a certainty, due to problems in comparability of cause-of-death statistics. In the past, the trend in COPD mortality has been upwards. Over the short time period of 1980 to 1985, there have been substantial declines in death rates in most countries for the major causes of death, but not for COPD or lung cancer. It is not clear, however, whether COPD mortality is continuing to increase everywhere. In only 9 countries is the trend upwards and only in women above age 55 and in men above age 75. In the other 22 countries, trends are not clearly upwards or downwards. Longer time-trend statistics are needed to corroborate and explain these patterns and the apparent intercountry differences.

Morbidity and Mortality from Chronic Obstructive Pulmonary Disease—J Manfreda, Y Mao, W Litven. Am Rev Respir Dis 1989;140: S19.

The National Mortality Database of Statistics Canada was used to study chronic obstructive pulmonary disease (COPD) mortality trends between 1950 and 1984 by age, sex, birth cohort, and province. Provincial hospital statistics were combined for the period 1971 to 1984 to determine age-specific hospital separation rates for COPD. Utilization of physician services for COPD was determined for one province using physicians' claims for payment. Results of these analyses indicate that mortality from COPD has leveled off for men and is increasing for women; the trend is different for combined chronic bronchitis—emphysema and for asthma. Mortality from COPD varies by age, sex, birth cohort, and province. Mortality from COPD stopped increasing with the 1910 to 1914 birth cohort in men and the 1920 to 1924 in women. Male and female birth cohorts born after 1920 to 1924 have decreased mortality. It is speculated that the age-standardized mortality will decrease in the near future. Surveys of smoking habits over time support the hypothesis that a proportion of sex, birth cohort, regional, and socioeconomic variation of COPD mortality is due to smoking. Hospital morbidity is higher for men than for women, particularly for those more than 65 y of age. Between 1971 and 1983, mortality/morbidity ratios were relatively stable. Asthma mortality and morbidity are increasing; changing concepts about the disease may be one reason for this observation.

Childhood Near-Drowning: Is Cardiopulmonary Resuscitation Always Indicated—MA Nichter, PB Everett. Crit Care Med 1989;17:993.

The case histories of 93 consecutive pediatric near-drowning victims admitted to All Children's Hospital from 1983 to 1987 were retrospectively reviewed for patient status on ED arrival and eventual outcome. Age, sex, length of submersion, core temperature, pH_a, absence of spontaneous respiration, lack of response to pain, and pupillary nonreactivity were all found to be unreliable predictors of outcome. Of those who received CPR, 68% went on to intact survival. The use of cardiotonic medicines to establish a perfusing cardiac rhythm in the initial resuscitation was associated with an eventual outcome of severe neurologic damage or death in all instances. The decision to use cardiotonic medicines in the resuscitation of pediatric near-drowning victims should be weighed carefully against their ultimate chances of intact survival.

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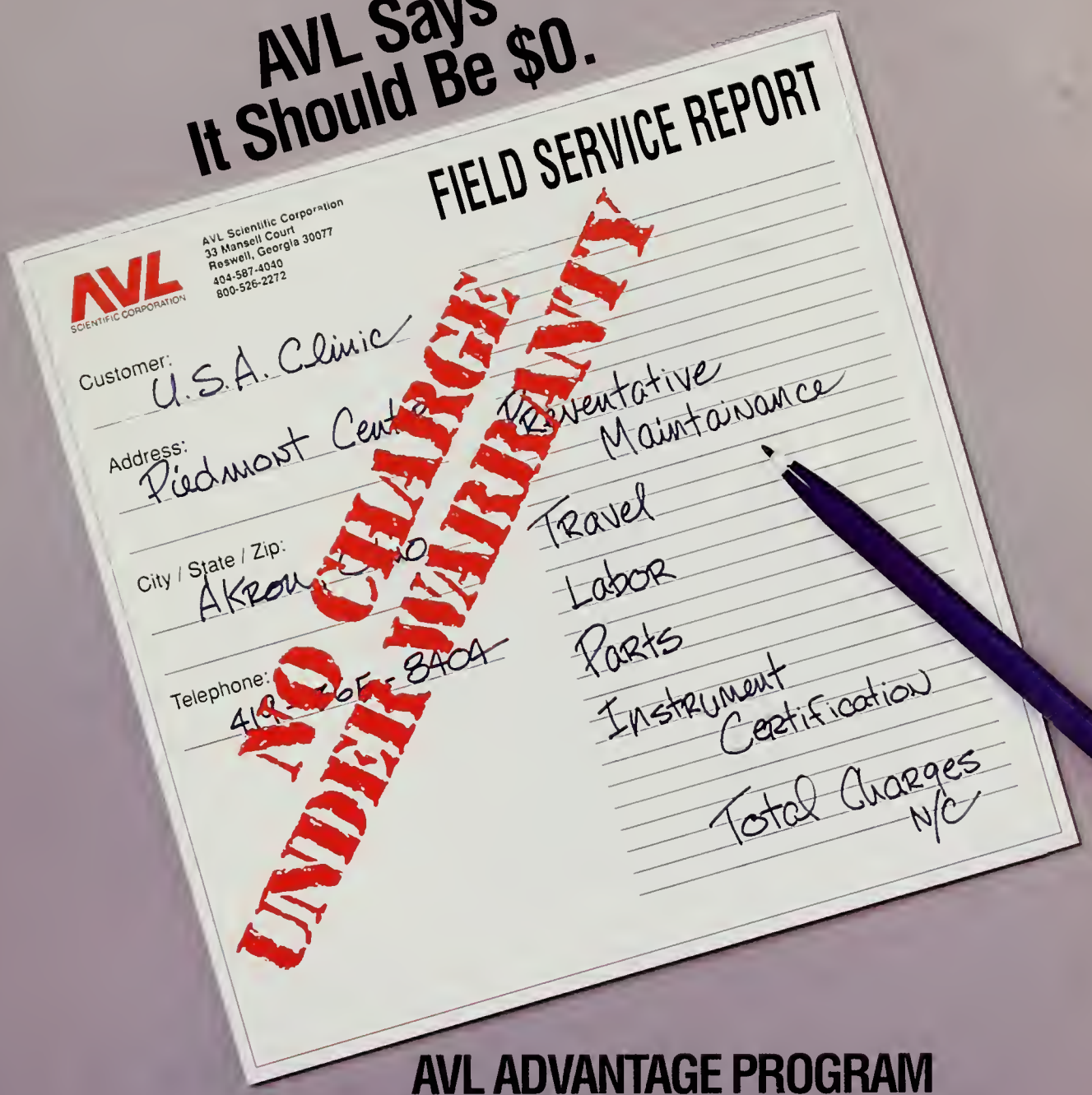

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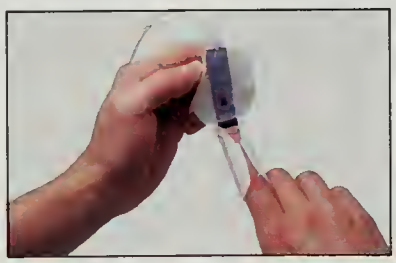

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Wanted: Manuscript Reviewers for RESPIRATORY CARE

Like other responsible professional journals, RESPIRATORY CARE puts manuscripts through peer review. That is, some of the author's peers (colleagues) examine the manuscript and advise the editors on its usefulness, accuracy, completeness, and clarity. This is a blind process: The reviewers don't know who wrote the paper, and the author won't be told who reviewed it. The peer review of a paper is designed to assure that the scientific literature is written to high standards, and reviewers play a vital role in the creation of a journal such as RESPIRATORY CARE.

This journal has a pool of manuscript reviewers, but the editors would like to enlarge it—tapping more talent, allowing others to participate. Therefore, in this editorial we invite readers to volunteer to review manuscripts. If that idea interests you, read on—whether you are a therapist, technician, technologist, physician, or engineer.

Who Can Be a Reviewer?

Reviewers should have true experience in the topics they review—and be familiar with the relevant literature. Take the topic of high-frequency ventilation, for example. The reviewer we want for this topic has both read the publications about high-frequency ventilation *and* employed it in his practice. It is not enough to be interested in this kind of ventilation and to have read about it. The reviewer should have had hands-on experience. Thus, in order to sign up for the reviewer pool, you must tell us which respiratory care topics—general and/or specific—you consider yourself really on top of. By the way, reviewers don't have to be experts in grammar and all that. Their function is to look at a manuscript's *content*.

What Are the Topics?

You may consider yourself competent in a broad-based, general topic, such as adult critical care, adult noncritical care, education, pediatrics, pulmonary function testing, neonatology, management, rehabilitation, home care, or others.

Taking the narrower, more specific view, your area may be auto-PEEP, statistics, CPR, CO_2 monitoring, Reye's syndrome, intracranial pressure, sleep apnea, hemodynamic monitoring, pulse oximetry, exercise testing, cystic fibrosis, CAI, infection control, sputum induction, pneumonia, IMV, pressure-support ventilation, weaning from ventilator support, tracheal tube cuffs, research design, ECMO, surfactant treatment, or any of many, many others. For a long list to pick from, examine the headings in the 10-year subject index found elsewhere in this issue of RESPIRATORY CARE.

How Can You Sign Up?

If you have expertise to bring to manuscript reviewing, and if you'd like to join the reviewer pool, send us a brief resume. Please prepare it in the format seen in this editorial's appendix. That is, first list your name and credential letters, professional address, and daytime telephone number. Then list the topics you could review—general topics, specific topics, or a mix—up to *six* of them altogether, not more. Finally, provide basic information about your professional education and experience.

What Will Happen Then?

Your name and topics will be carried in our reviewer files. If pertinent manuscripts are up for

review, we will try to send you assignments. I say "try" because if a given manuscript is about CPAP, for instance, and that's a topic you listed, but 150 others have listed it too, your name may or may not be pulled from the hat.

Reviewer Aids

If you receive a manuscript to review, it will be accompanied by instructions. For several kinds of papers, we have developed reviewer's kits. The kits organize the reviewer's task, trying to make sure that all the pertinent aspects of the manuscript are evaluated. So—if you are new at the game, there will be a guiding hand.

In this connection, I recommend that reviewers—current and future—obtain the inexpensive paperback book, *Interpreting the Medical Literature,* 2nd edition, by Stephen H Gehlbach MD. This book is intended to help clinicians read and understand research articles. I have found it to be clearly written and a good entrée to the world of research, case reporting, measurement-making, statistical analysis, interpretation, and all the rest of an environment that may not be familiar. I recommend it particularly to reviewers—but researchers, authors, and anyone wishing to continue his or her education should benefit from a few hours invested in this book's pages.

For Your Info

If, as might happen, hundreds sign up for the reviewer pool, we won't be able to acknowledge receiving your resume. Not to worry—put your return address on the envelope and trust the post office.

An important part of reviewing manuscripts is promptness. Authors anxiously await the results of our evaluation, and we ask reviewers to complete their work and mail their review back to us within two weeks. Thus, would-be reviewers should be well organized and willing to put the manuscript for review near the top of their To-Do stack.

Every year, in the December issue of RESPIRATORY CARE, we print a list of that year's reviewers, to thank them and to let their colleagues know who's been laboring behind the scenes so this journal can be as good as it is. That listing is a small part of a reviewer's reward. The larger part is the knowledge that the reviewer has performed an important professional task. Many of the same names have appeared in the December thank-you list year after year. Those persons consider reviewing to be a professional obligation and privilege. Some even say it's fun.

From Reviewing Manuscripts to Writing Them—Why Not?

We have found that successful authors often make good reviewers. Well, why not the other way round? Reviewers who learn to take apart the manuscripts of others, seeing what makes them tick, should then be better prepared to become successful authors themselves. Hey! This might happen to you.

If you're interested, mail your resume to:

Reviewer Pool
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Box 29686
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Phil Kittredge RRT
Adjunct Editor

APPENDIX

MODEL FOR A
MANUSCRIPT-REVIEW CANDIDATE'S RESUME

(Please follow this format)

Leslie Hotshot Therapist RRT
Respiratory Care Department
Pretty Good Medical Center
111 One Way Street
Bandagetown, Tennezona 22222
Tel: 123/456-7890

MANUSCRIPT-REVIEW TOPICS:

Adult Critical Care
High Frequency Oscillation
Leech Therapy for Asthma
Patient Positioning
Smoking Cessation

EDUCATION:

Graduated from Bandagetown Community College with an Associate's degree in 1981. Pursuing Bachelor's degree from Tennezona State University.

CREDENTIALS:

CRTT 1982, RRT 1984

POSITIONS & EXPERIENCE:

Staff Therapist 1981-85
Shift Supervisor 1986-present
In-service Educator 1988-present

OF Again—On Again!

What consists of two pages of fine print buried in the back pages of the January through May issues of RESPIRATORY CARE? You don't need to be an aficionado of Trivial Pursuit to come up with the answer to that one—the answer is the Open Forum Call for Abstracts, of course.

Now, about that fine print in the Call—you say, Do I really need to read it? Well, only if you're going to participate, only if your thinking of participating, only if you're hoping in your secret heart that a group from your institution (including you) will participate.

The Call may look dull, and the directions may seem tedious, but the Journal's Editorial Board and staff view the Open Forum with a sort of evangelistic fervor—so, that makes the Call for Abstracts a gospel of sorts.

We view the Forum as an opportunity for clinicians, managers, and educators to present the results of their studies, evaluations, and observations. The Forum is the fourth step in a natural, logical progression that can culminate in the publication of a paper.

What is Step 1? As with almost any endeavor, Step 1 is, of course, expressing the IDEA—What's to be studied, evaluated, reported?

Next comes Step 2—plotting and planning, choosing the design, expressing the hypotheses, determining the methods to be used. How will subjects be enrolled and how many will be needed or how many units or models will be tested? What statistical analysis will be employed?

Step 3 is doing the work according to plan—with good records and complete information.

Step 4—writing and submitting the abstract—is relatively easy if Steps 1-3 have been done correctly. The abstract almost writes itself: Introduction, Materials and Methods, Results, Conclusions; or Identification of method or device, Evaluation Method, Results, Experience, Conclusions; or for a Case Report, the case summary and explanation of the significance of the case. However, the time and thought investment must have been made 'up front'—in the planning, design, and implementation.

Step 5 is making a presentation in the Open Forum based on the abstract, and Step 6 is submitting a paper for publication based on the abstract and presentation. We won't elaborate on those now.

What gives the newcomer to clinical investigation and equipment or method evaluation the biggest headache? Where do many go wrong? The answer to that probably is: in the design and statistical analysis. The moral of that story is Get help at the planning stage. Don't wait until your data are in.

A special word to the would-be reporter of an interesting case—Be sure that you have the necessary information to make your case 'come alive' for the reader—a chart complete enough to allow the story to unfold and to defend the course of action that was chosen—the important x-rays, laboratory results, photographs of bronchoscopy findings, or whatever. No matter how good the potential teaching value of a case, if records are incomplete, the case may never be acceptable for presentation.

What's the most common complaint of would-be abstract submitters?—Not enough room to explain the findings and the data. The solution to that problem goes back to planning and organization—addressing manageable, answerable questions in the most direct way possible.

To make this all a little easier, read the Call for Abstracts two or three times before you start to work and do some other reading, too—for example, the papers by Pierson (Respir Care 1982;27:29-32), Hess (Respir Care 1986;31:766-767), and Kittredge (Respir Care 1985;30:1057-1061) reprinted in the

Journal's Author and Typist's Kit (available from the Editorial office), the books *Fundamentals of Respiratory Care Research,* by Chatburn and Craig (Norwalk CT: Appleton & Lange, 1988), and *Interpreting the Medical Literature,* by Gelbach (New York: MacMillan, 1988).

And, finally, remember that the Open Forum is not a competition. Each abstract is peer-reviewed by a panel of six content-expert investigators. Each abstract is judged on its own merit. No predetermined number of abstracts must be accepted or rejected. Each year, a number of first-time investigators have their work accepted for presentation in the Open Forum. So, the OF is on again!

Pat Brougher
Editor

Respiratory Care in the Era of Preventive Medicine

Ronald B George MD

Introduction

Since the 1965 amendment of the social security laws, which created the Medicare system of government-supported health care, the government has assumed an increasing role in reimbursement of medical expenses for the elderly, a large percentage of whom have chronic respiratory diseases. Over 22 million Americans currently suffer from chronic obstructive pulmonary disease (COPD), with a marked increase in prevalence over the past two decades.[1] The incidence of chronic bronchitis has increased 52% and that of emphysema 32% during this time. COPD now ranks fifth among the leading causes of death in Americans, accounting for 3.4% of all deaths in 1985. Mortality trends suggest that age-adjusted mortality from COPD is increasing (a rise of 21% from 1979 to 1984), in contrast to a declining mortality rate from other leading causes of death such as heart and cerebrovascular disease.[1] The economic impact of COPD in the United States is striking. The total cost of lung disease in 1986 was $40.9 billion, with COPD being responsible for approximately one fourth of this cost.

Among the leading causes of death and disability in our country (Table 1), four of the top five are smoking related.[2] Lifestyle also affects others among the top ten; for instance, obesity and exercise play a role in heart disease, cerebrovascular disease, and atherosclerosis, while alcohol and drug abuse affect the incidence of accidents, pneumonia, chronic liver disease, and suicide. Because the majority of health care costs today are met by third-party payers, federal and state governments and private insurance companies have identified prevention of chronic lung diseases as a major goal. It is cheaper to prevent serious diseases, especially those that are chronic, than it is to treat them once they have occurred. Public awareness of health care measures has also heightened the government's interest in cigarette smoking, drugs, alcohol, nutrition, and exercise. I will address the effects of these changes in attitudes on the respiratory care profession.

Table 1. The Ten Most Common Causes of Death in 1980

Cause	Number of Deaths
1. Diseases of the heart	763,060
2. Malignant neoplasms	414,320
3. Cerebrovascular disease	170,420
4. Accidents and adverse effects	106,550
5. Chronic obstructive pulmonary disease and allied conditions	55,810
6. Pneumonia and influenza	55,720
7. Diabetes mellitus	34,230
8. Chronic liver disease and cirrhosis	31,220
9. Atherosclerosis	29,830
10. Suicide	28,290

Reprinted, with permission, from Lenfant C. Lung research: Government and community. Am Rev Respir Dis 1982;126:753.

Dr George is Professor and Deputy Head, Department of Medicine, LSU School of Medicine, and Medical Director, Respiratory Care, LSU Medical Center, Shreveport, Louisiana.

A version of this paper was presented by Dr George as the Program Committee Lecture at the Annual Meeting of the American Association for Respiratory Care, in Anaheim, California, December 4, 1989.

Growth and Development of Respiratory Care

The growth curve of the respiratory care profession has not been a straight line but rather a parabolic curve with slow early growth followed by a rapid growth phase in the 1960s, 70s and 80s (Fig. 1).

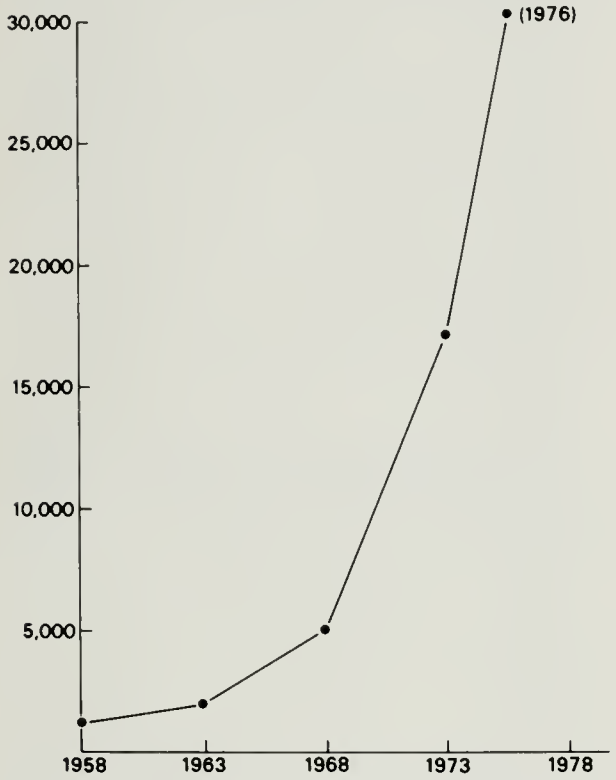

Fig. 1. Growth in membership of the American Association for Respiratory Therapy, 1958-1976, Reprinted, with permission, from Burton GG, Gee GN, Hodgkin JE, eds. Respiratory Care. 1st ed. Philadelphia: JB Lippincott Co, 1977:9.

The development of the field can be divided into four phases, as shown in Table 2. At the end of World War II, respiratory care consisted primarily of supplemental oxygen (administered by "oxygen technicians") and surgical anesthesia. The poliomyelitis epidemics that occurred before the advent of the vaccines in the mid-50s provided an impetus for development of long-term mechanical ventilation. Early ventilators like the Drinker iron lung and the first practical positive-pressure ventilator, the Mörch piston ventilator, were used during the 1940s and 50s.

The rapid growth of respiratory care began in the late 1950s and early 60s with the development of reliable arterial blood gas analyzers, followed by pressure-cycled ventilators, and later the modern volume-cycled ventilators. During this period of rapid growth, respiratory therapists were in great demand, and respiratory therapy departments were established in many hospitals. Intensive care units emerged, with a variety of monitoring devices that made the care of ventilator-dependent patients both more effective and more personnel intensive.

The rapid growth of respiratory care in this country coincided with a rapid increase in health care costs, so that health care became a major component of our total gross national product. Thus, as the Tax Equity and Fiscal Responsibility Act (TEFRA) was debated and passed to change the reimbursement process to prospective payment in October 1983, respiratory care was singled out as one of the areas for intensive study in cost control.[3,4] Respiratory care departments changed from income-generating units to expenditure units in hospitals, and a period of cost containment was initiated that has continued to the present time. The specialty has continued its gradual but continued growth by adapting to new medical knowledge; for example, IPPB has been replaced by chest physical therapy as a mode of

Table 2. The Four Eras of Respiratory Care

The Early Days (prior to 1960)
Oxygen therapy
Surgical anesthesia
Early assisted-ventilation techniques
The Rapid Growth Phase (1960-1983)
IPPB
Arterial blood gas analysis
Modern mechanical ventilators
Respiratory therapy departments
Intensive care units
Monitoring devices
The Era of DRGs (1983-present)
Cost-saving measures
Diversification
The Future (1990 and beyond)
Preventive techniques
Patient education
Home care and rehabilitation

improving bronchial mucus clearance, and postoperative respiratory care has been tailored more to fit the needs of specific patients—with resulting savings in hospital costs.[5,6] Attempts to move long-term ventilator-dependent patients from high-cost intensive care units to non-hospital settings including the home have increased.[7,8] During the so-called era of DRGs, diversification has been a rational response to limited reimbursement and has allowed continued growth of the profession (Table 3).[9] Educational programs have changed from respiratory therapy programs to departments of cardiopulmonary science to train students to respond to the needs of a changing profession.

Table 3. Most Common Non-Respiratory Responsibilities (79% of 657 Respiratory Care Departments)[9]

Health Promotion and Education
Noninvasive Cardiac Diagnostics
Electrocardiography
Echocardiography
Cardiopulmonary Rehabilitation
Home Care
Electroencephalography (EEG)

Current Status of Respiratory Care

Respiratory care's ability to adapt rapidly to a changing environment results from its strong national organization, the American Association for Respiratory Care, and to continued dynamic leadership. It also has resulted from the strong support of physicians over the years. This ability to change will be valuable in adjusting to the current alteration in emphasis from intervention to prevention of chronic diseases. Some of the areas in which respiratory care practitioners should become increasingly involved are listed in Table 4.

Substance Abuse Prevention and Cessation

Cigarette smoking has been identified by the U.S. Public Health Service as the most important avoidable cause of death and disability in the United States.[10] Although the number of smokers has decreased since the first Surgeon General's report on smoking and health in 1964, a 1984 survey estimated that approximately 30% of all adults in this country still smoke.[11] The health costs attributable to smoking are estimated at $16 to $17 billion a year; and when loss of productivity and direct medical costs are added, the total may exceed $41 billion per year, or $200 per capita.[11–13] Approximately 10-15% of cigarette smokers will develop significant impairment of lung function.[14] It is estimated that 80-90% of deaths from COPD in this country in 1983 were related to smoking and thus were preventable.[15] In smokers who develop COPD, the rate of decline in the forced expiratory volume in the first second (FEV_1) is increased from the 30-45 mL per year seen in normal non-smokers to 80-100 mL per year.[16] COPD patients who continue to smoke eventually develop severe obstructive airways disease if they survive long enough. In long-term cigarette smokers who do not die of respiratory failure, the risk of developing cancer of the lungs is significantly increased. Cigarette smoking has also been identified as a risk factor for other chronic illnesses including cardiovascular diseases, and in this country the overall mortality ratio for adult cigarette smokers vs non-smokers is about 1.7.[11]

Table 4. Preventive Strategies in Respiratory Care: A Plan for the 1990s

Substance Abuse
Smoking cessation
Alcohol
Drugs
Patient Education
Use of inhalers and nebulizers
Nutrition
Weight reduction
Rehabilitation
Physical therapy
Exercise
Vocational and communications skills
Patient monitoring
Prophylactic Aerosols
Pentamidine
Artificial surfactant
Antibiotics in ICU patients
Antiviral agents (interferon)

Table 5. Fagerström Nicotine Dependency Test[20]

	A = 0	B = 1	C = 2
1. How soon after you wake up do you smoke your first cigarette?	After 30 min	Within 30 min	—
2. Do you find it difficult to refrain from smoking in places where it is forbidden, such as the library, theater, doctor's office?	No	Yes	—
3. Which of all the cigarettes you smoke in a day is the most satisfying?	Any other than the first one	The first one in the morning	—
4. How many cigarettes a day do you smoke?	1-15	16-25	More than 26
5. Do you smoke more during the morning than during the rest of the day?	No	Yes	—
6. Do you smoke when you are so ill that you are in bed most of the day?	No	Yes	—
7. Does the brand you smoke have a low, medium, or high nicotine content?	Low	Medium	High
8. How often do you inhale the smoke from your cigarette?	Never	Sometimes	Always

How to interpret your score:

Scores of 7 or higher: highly dependent on nicotine.
Scores of 6 or less: a low-to-moderate nicotine dependence.

Nicotine has been shown to meet all the criteria of a physically addicting substance.[17] The difficulty in quitting and the relapse rate over time experienced by smokers suggests that cigarette smoking is as addicting as alcohol and narcotics, the major addicting substances in our society.[18,19] The degree of nicotine dependence can be determined simply but with fair reliability by the Fagerström questionnaire, shown in Table 5.[20] Packets of these forms for office use are available free of charge from Lakeside Pharmaceuticals, Division of Merrell Dow Pharmaceuticals Inc, Cincinnati OH 45215. Patients with total scores of 7 and above have a high degree of physical dependence and may be candidates for replacement therapy with nicotine chewing gum or for measures that decrease dependence, including sedatives, tranquilizers, or clonidine patches.[21]

Outpatient smoking cessation programs are ideal for physician-therapist interaction. A prototype for an office-based smoking cessation program that uses self-help booklets such as "Freedom from Smoking in Twenty Days" and "A Lifetime of Freedom from Smoking," available through the American Lung Association, is shown in Table 6. Billing is possible for smoking cessation visits provided they are made a part of the regular office visits for management of COPD. Serial monitoring may be performed at the same time with portable spirometry or peak flow meters, and theophylline blood levels can be tested by the therapist in the office with one of the commercially available finger stick methods that yield quick results. Validation of smoking behavior by analysis of expired carbon monoxide is another simple and useful office procedure. The input of a nonphysician in a smoking cessation program has been shown objectively to be a valuable addition resulting in a significant increase in successful cessation.[22]

New Preventive Methods for Respiratory Infections

For many years, attempts have been made to prevent or treat respiratory infections with inhaled antibiotic mixtures. Success had been limited,

Table 6. Steps in an Outpatient Smoking Cessation Program Incorporating Physician-Therapist-Patient Interaction

	Physician	Therapist
Step 1:	Explains health effects of smoking Obtains patient agreement to enter program	Shows a film about smoking and cessation methods Contracts with patient to quit smoking Receives registration fee (optional) Administers questionnaire to assess nicotine dependence
Step 2:	Prescribes nicotine gum or clonidine for highly dependent patients	Supplies self-help booklet (eg, "Freedom from Smoking in 20 Days") Conducts weekly group therapy sessions for up to 8 weeks
Step 3:	Schedules office visits (monthly at first, then at 3-month intervals for 1 year) Prescribes nicotine gum or clonidine as indicated	Administers questionnaire (related to benefits, problems, stressful situations) Provides objective testing (expired CO analysis, urine or salivary cotinine) Provides self-help booklet (eg, "A Lifetime of Freedom from Smoking")

however, until the recent epidemic of the acquired immunodeficiency syndrome (AIDS) and the demonstration that inhaled pentamidine can markedly reduce the relapse rate with pneumocystis infections,[23] caused a resurgence of interest in this technique. A jet nebulizer with a filter on the exhalation port is used to administer the aerosol at monthly intervals. The technique has been shown in pilot studies to be safe and effective in preventing relapse following an initial episode of pneumocystis pneumonia.[23] Prophylaxis for nosocomial gram-negative lower respiratory-tract infections using locally applied or aerosolized non-absorbable broad-spectrum antibiotics has recently been effective in severely ill ICU patients.[24] Trials are currently underway with combinations of such non-absorbable antibiotics as polymyxin B, gentamicin, and amphotericin B, applied locally to the oropharynx to prevent colonization. A decrease in colonization has been shown to decrease aspiration of these organisms into the lung periphery and thereby to decrease the development of nosocomial pneumonias.[24] Other applications of inhaled antimicrobial agents are currently being evaluated.

Other Areas of Investigation

Inhaled aerosols may also be useful in the management of other potentially fatal illnesses including the infant respiratory distress syndrome. In one study, surfactant aerosols were administered to 35 premature infants in a neonatal ICU. Calf-lung surfactant extract delivered by aerosol resulted in a 56% decrease in the alveolar-arterial oxygen ratio in treated vs control patients.[25] This therapy may also be useful in adult patients with ARDS, in whom an abnormality of surfactant function has been demonstrated.

Home care for patients with severe chronic lung disease, including those who are ventilator-dependent, is expanding, and a whole new field of home care is developing rapidly. Much of the future of respiratory care appears to be in non-hospital based practices, as has occurred in the past with such specialties as physical and speech therapy; the special expertise of respiratory therapists for ventilator management should prove useful in the home care environment of ventilator-dependent patients. An expansion of the therapist's role in the home might include such areas as vocational rehabilitation, improvement in the communications skills of ventilated patients, and nutritional management.

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World Congress on Home Care

Robert M Kacmarek PhD RRT and David J Pierson MD

The World Congress on Home Care, cosponsored by the American Association for Respiratory Care, was held on March 14-18, 1989, in Rome, Italy. This Congress was the first devoted to home care. Previous Congresses in Denver, Colorado, in 1987 and 1988 and in Lyon, France, in 1989 had addressed specific aspects of home care (eg, oxygen therapy and mechanical ventilation) but had not addressed the broader issues that affect technology assisted persons at home or the home care issues that center on respiration, nutrition, oncology, nephrology, and geriatrics. About 650 persons from some 50 countries representing every continent except Antarctica attended the meeting.

The U.S. respiratory care community was well represented. Your reporters served on the Planning Committee. Patrick Dunne MBA RRT presented one of the keynote lectures "Home Care Services Organizations" and Mary Gilmartin RN RRT lectured on "Care Givers in Chronic Respiratory Failure" and Patient Selection for Mechanical Ventilation." Other lectures included "Respiratory Home Care in the USA" and "Pulse Oximetry for Patient Surveillance during Long Term Oxygen Therapy" by David Pierson and "Noninvasive Mechanical Ventilation" by Robert Kacmarek. Sue Sortor RRT conducted a workshop on "Noninvasive Positive Pressure Ventilation." Paul J Mathews MPA RRT and Ray Masferrer RRT were official representatives of the AARC. The respiratory section of the meeting had the largest attendance, and its program spanned all days of the meeting.

Issues related to the development of home care organizations, national home care policies, and problems related to funding were addressed on a global level. Attendees concurred that AIDS would become a driving force in the home care arena during the 1990s. Topics presented during the respiratory sessions covered the total scope of home respiratory care from oxygen and aerosolized pharmacologic therapy to the management of patients with chronic ventilatory failure and sleep disturbances. Much attention was given to noninvasive approaches to ventilatory support and to the long-term effects of oxygen therapy. Overriding issues were patient selection and the expanded use of elective approaches to ventilatory support. Participants agreed that the number of persons utilizing home respiratory care is like to increase markedly in the 1990s.

A large part of the Congress was dedicated to the presentation of original work. A total of 208 abstracts were presented including 60 in oncology, 25 in nephrology, and 24 in nutrition. The largest single group of abstracts (67) addressed respiratory home care. Twenty one of those abstracts are reproduced in this report exactly as they were submitted and presented at the meeting. These abstracts reflect respiratory home care activities in 13 countries outside of the USA and were selected as most representative of the original work presented in the respiratory section of the meeting. Some of these abstracts are primarily descriptive in nature portraying patient care activities in the various countries, but others follow rigorous scientific methodology. These abstracts document the fact that we in the United States are not alone in attempting to meet the challenges of home care for our chronically ill, but rather are a part of an increasingly well-developed world system addressing home care issues.

The rapid development of home care world wide was emphasized by the founding of the International Home Care Association (IHCA). This organization's charter stresses as its goals the development of home care in all disciplines on a global basis and includes in its membership persons involved in every aspect of home care: "to include, but not limited to health care providers, manufacturers and distributors." Three members of the AARC, Patrick Dunne, David J Pierson, and Robert Kacmarek are listed as charter members of the organization, with David J Pierson being named President-Elect and Patrick Dunne Treasurer. Future Congresses on International Home Care have been planned for Italy in the Spring of 1991 and Seattle in the Summer of 1992.

For us, this meeting was academically rewarding and intellectually stimulating. We were encouraged by the magnitude of global interest in home care and found the activities surrounding this meeting a testament to the increasing opportunities for respiratory care practitioners in the world market.

Dr Kacmarek is Assistant Professor, Department of Anesthesia, Harvard Medical School, and Director of Respiratory Care Services, Massachusetts General Hospital—Boston, Massachusetts. Dr Pierson is Professor of Medicine, University of Washington, and Medical Director, Respiratory Care Department, Harborview Medical Center—Seattle, Washington.

SELECTED ABSTRACTS FROM THE WORLD CONGRESS ON HOME CARE

Held in Rome, Italy
March 14-18, 1989

UNDER THE PATRONAGE OF

Ministero della Sanità della Repubblica Italiana
Assessorato alla Sanità della Regione Lazio
Assessorato alla Sanità della Provincia di Roma

UNDER THE AUSPICES OF

Hospitaller Order of St. John of God
Fatebenefratelli International Foundation
American Association For Respiratory Care
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HOME CARE AND REHABILITATION

ITALY

PULMONARY REHABILITATION AND PHYSICAL TRAINING IN PATIENTS WITH CHRONIC OBSTRUCTIVE LUNG DISEASE (COLD): A CONTROLLED STUDY.

S.Scalvini, K.Foglio, S.Marangoni, M. Volterrani, M. Schena, A.Dotti.
FONDAZIONE CLINICA DEL LAVORO - Dpt. of Medical Rehabilitation Via Pinidolo,23 - 25064 Gussago Brescia Italy.

A controlled study on the effects of exercise training in 47 COLD patients was performed. 37 patients underwent a general rehabilitation program and a 4 weeks training on a cycle ergometer at the 70% of their maximum work capacity (trained group) and 10 patients underwent only a pulmonary rehabilitation program for the same time (control group). Respiratory function tests and arterial blood gas analysis at rest, exercise capacity, pulmonary and cardiac parameters during an effort test were recorded before and after the program. At the same load reached in the first effort test the control group showed no differences in the parameters before and after the program. Trained group showed a statistically significant decrease in heart rate (HR) ($p < 0.0025$), blood pressure (BP) ($p < 0.05$), respiratory rate (RR) ($p < 0.0001$), ventilation (VE) ($p < 0.0001$). At the maximum load reached in the second effort test, an increase in HR, VE, oxygen consumption ($\dot{V}O2$), Weber index, anaerobic threshold (AT), work capacity ($p < 0.0005$) and RR ($p < 0.0125$) was recorded; while in the control group an increase in AT ($p < 0.05$) and work capacity ($p < 0.05$) was recorded.

In conclusion to achieve improvement in performance, exercise tolerance, and in some cardiac and pulmonary parameters, an exercise training is required, in COLD patients, during a general rehabilitation program.

FRANCE

SURVIVAL OF PATIENTS REFERREO TO A FRENCH REGIONAL ASSOCIATION, ADEP-ASSISTANCE, FOR HOME CARE RESPIRATORY ASSISTANCE

D. Elkharrat, J M Bédicam.
ADEP- Assistance - 92380 Puteaux - France

French Regional Associations constitute an appropriate observatory for the evaluation of various aspects of Home Care Respiratory Assistance (HCRA) Our purpose is to describe, through one of these associations, ADEP-Assistance, for the year 1987 the survival of patients according to the aetiology of their respiratory handicap (RH). As of december 1986, there were 1485 patients registered with ADEP, mean age 49 1 years, 66 % males. The aetiology of their RH is a pulmonary disease (P) in 64 3 % of cases, neuromuscular (NM) in 23.2 %, a chest-wall disease (CW) in 4 3 %, a laryngeal or tracheal disease (LT) in 2.8 % and bronchodysplasia in 1 7%. The main techniques of HCRA were, ventilation through tracheostomy (VT) in 31 % of patients, through a mouth-piece (VM) in 18 % or a nasal mask (VN) in 3 % , 62 % had oxygen therapy alone or in combination with the other techniques. In 1987, 707 new patients, mean age 50 2 years, 68.6 % males, were referred to ADEP for HCRA. There were 97 children under the age of 16 The main underlying diseases to the RH were P 66.5 % and NM 13.7 %

During the same year the outflow of patients equaled 456. In 77 % of patients death was the cause for interruption of HCRA, in 9 3 % HCRA was not required any more and in 13 % HCRA was interrupted for various reasons. 1)Patients who required no more HCRA were originally admitted for a P disease in 34 3 % of cases, bronchodysplasia in 23 %, NM (mainly myasthenia gravis or a demyelinating polyneuropathy) in 17 2 %, LT in 14 3 %, miscellaneous 11 2 %. 2) Indications of HCRA in the deceased patients were P 74 %, NM 7 5 %, CW 1 3 %, LT 3.2 % , 14 % of patients had a malignant pathology, either of the lung or with a secondary extension to the lung Oxygen was the main treatment in 95% of the P patients, whereas 50 % of NM patients were ventilated through tracheostomy, 36 % through VM and 1 % VN When patients with cancer are substracted it is noted that over 50 % of P patients died within 12 months following their admission at ADEP, whereas only 28 % of NM patients died in that time-limit.

Conclusions 1) These data are consistent, in terms of survival, with the established notion that HCRA is more beneficial in the respiratory handicap of neuromuscular origin. 2) However, the high mortality in the P group in such a short time- span following HCRA, calls for clarification

FRANCE

LOW VENTILATORY RESPONSE TO CO2 REBREATHING IN CHRONIC NEUROMUSCULAR AND KYPHOSCOLIOSIS SETTINGS

J B Vercken*, Cl Fromageot**, J C Raphael*, D Elkharrat*, J de Lattre**, Ph Gaidos*
*Service de Réanimation Médicale,**Service des Explorations Fonctionnelles Respiratoires - Hôpital Raymond Poincaré - 92380 GARCHES FRANCE

Decreased ventilatory responses to CO2 rebreathing have been reported in chronic neuromuscular affections with respiratory handicap Our purpose was to investigate these data and attempt to progress in comprehension of underlying mechanism

Patients methods We studied 16 adult patients 8 with myopathy (N° 1 to 8) (3 Ouchenne's muscular dystrophy, 1 maltase acid deficit, 2 Steinert's myopathy 2 of unknown origin), 6 with kyphoscoliosis, (N° 9 to 14), 2 with myasthenia gravis, (N° 15 and 16) All hed a respiratory handicap moderate to severe (vital capacity above 50% only for N° 4,7,13,15 and 16, and as low as 8% for N°2 and 3) All patients were studied for ventilatory response ($\dot{V}$) to CO2 rebreathing. Alveolar and tidal CO2 pressure (PAETCO2) served as index of CO2 stimulus Arterial blood gases at rest demonstrated normocapnia (N° 1,3,4,6,8,13,14,16), moderate (44 torrs, N° 9 and 11) or marked hypercapnia (47 to 56 torrs) Only 8 patients were apt to exercise, on a bicycle (N°4,5,6,7,9,13,14,16) with arms (N° 9 and 16) or legs. They developed a power ranging from 50%-80% of their maximal oxygen consumption ($\dot{V}O2$ max)

Results Patient's slope of ventilatory response versus PAETCO2 was low of very low . respectively, from N° 1 to 16, $\dot{V}$/PAETCO2 equal : 0 20 - 0 29 - 0 24 - 0 67 - 0 - 0 16 - 0 39 - 0 13 - 0 57 - 0 77 - 0 30 - 0, - 0 45 - 0 69 - 0 16 - 0 98 expressed in l/min/mmHg Reported values in healthy subjects, widespreaded, usually range from 1 to 3 99 l/min/mmHg Our laboratory controls (12 subjects - some of them tested two or three times) ranged from 0 74 to 3 29 l/min/mmHg. In the 8 patients able to exercice, the slope of ventilatory response as a function of oxygen consumption ($\dot{V}/\dot{V}O2$) was similar or superior to normal. Exercise ventilation was, for all of them, markedly greater than the one reached at end of their CO2 rebreathing test.

Conclusion. These results are not only in good agreement with previous findings of low CO2 ventilatory responses in patients with neuromuscular diseases, but also exhibit similar findings for the kyphoscoliosis patients studied. The fact that ventilatory response at exertion appears within or above normal range suggests the anomaly is related with respiratory control regulation, and not to inability of thoraco-pulmonar effector

CANADA

A UNIVERSITY HOSPITAL FUNDED RESPIRATORY HOME CARE PROGRAM. MODEL FOR TERTIARY CARE

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Respiratory Home Care originated as a project at the University of Alberta Hospital in 1974 to provide support for chronically ill respiratory patients. This support enables these patients to live outside of an institutional setting. From October, 1987 until October, 1988, 140 patients received care from this program. For this study, we have chosen to present the data on those patients requiring tracheostomies and/or ventilatory assistance. 20 were male and 15 were female with ages ranging from less than 1 year to 70 years with a mean age of 50 years. They had a variety of diseases; Poliomyelitis (24), Neuromuscular/skeletal (5), Congenital abnormalities (3), Sleep apnea (2), and C.O.P.D. (1). 30 had a ventilator, 23 had a tracheostomy, and 18 had both. 11 of these patients also required supplemental oxygen. Of these, 15 had pressure ventilators (Bantam, LP3, LP4), 12 had volume ventilators (PLV 100, Companion 2800), and 6 had rocking beds. 3 patients had used more than 1 ventilator system. The method of delivery of positive pressure consisted of 18 via tracheostomy tube (5 of whom required an inflated cuff), 5 mouthpieces, 2 nasal masks, 1 pneumobelt, and the remainder used only rocking beds. The fund required for equipment, health care at home, and general living expenses have come from employment, government social services, families, charitable organizations, insurance, pensions, or a combination of these. . We have been successful in helping these people live outside an institution along with community support systems. The mean hospital stay for these 35 patients over the past year has been 13.4 days with a range of 0 to 27 days. It has been cost effective to maintain ventilator and tracheostomy patients in the community. The actual per diem cost of an in-hospital patient is at least $300/day. The cost at home ranged from $45/day to $175/day depending upon the level of service, home help and equipment required. We estimated that the home care cost for these 35 patients was at least $2.5 million less per year than if they were to live in hospital. We have been successful in maintaining patients on long term ventilation at home, enabling them to participate more fully in the community with a major cost savings to society.

ASTHMA AT HOME

FINLAND

ASTHMATIC PATIENTS WANT TO MONITOR THEIR SYMPTOMS AND TO PERFORM PEAK EXPIRATORY FLOW MEASUREMENTS AT HOME.

Kava, T. Ounasrinne Hospital, Kolpeneentie 21, SF-96440 Rovaniemi, FINLAND

In asthma, the patient himself plays an important role in the management of the disease. Daily peak flow (PEF) measurements and records of asthmatic symptoms provide valuable data for the physician in assessing the severity of the disease and in planning therapy. The purpose of the present study was to investigate the readiness of patients to conduct PEF measurements and to keep a record of their symptoms at home. The subjects of the study comprised 40 men and 62 women, mean age 44.5 yrs (16-70 yrs), suffering from asthmatic symptoms. The patients were each given a mini-PEF meter for use at home, accompanied by both oral and written instructions for its use and for recording symptoms. Mean duration of the follow-up was 2.3 months (range 1 week - 5 months).

At the time of returning the meter, 83 subjects (81.4 %) filled in a questionnaire consisting of ten items concerning self-monitoring at home. 75 (90 %) of those who completed the questionnaire considered self-monitoring very useful or quite useful. According to 49 patients (59%), the self-monitoring caused no or very little inconvenience. 77 patients (93 %) experienced self-monitoring as meaningful or very meaningful, and 72 (87 %) as interesting or very interesting. Use of the flow meter and the recording of symptoms was considered easy or very ease by 78 (94 %) and 67 (81 %) patients, respectively. 76 patients (92 %) considered the monitoring of symptoms important or very important. From the point of quality of life, 61 patients (73 %) experienced the self-monitoring as less or much less negative than the disease itself. 56 patients (67 %) stated that self-monitoring helps patients to understand the nature of their symptoms and to cope with their asthma, and 48 patients (58 %) believed that monitoring makes patients realise the rationale of drug therapy. 57 (69 %) of the patients expressed a readiness to carry out periodical monitoring at home; 12 patients (14 %) were willing to perform continuous daily self-monitoring.

In conclusion, asthmatic patients were found to have a positive attitude toward self-monitoring of PEF and symptoms.

SWITZERLAND

AMBULANT CONTINUOUS INTRAVENOUS THERAPY WITH THEOPHYLLINE IN THREE PATIENTS WITH BRITTLE BRONCHIAL ASTHMA.

B. Speelberg, W.T.J. van den Brink, Dutch Asthma Centre Davos, Switzerland.

Asthma is called brittle when despite optimal treatment (including inhaled bronchodilators, theophylline, inhaled and oral corticosteroids), asthmatic attacks occur frequently and severely. In patients who are already using theophylline preparations, intravenous therapy with theophylline seems to have better results.

We investigated the role of ambulant continuous intravenous infusion with theophylline in patients with brittle bronchial asthma, by means of the CADD I-pump. The CADD I-pump is a portable infusion pump with a medication cassette of 100 ml. The pump can be programmed for flow, expressed in ml per 24 hours. The pump has several alarm systems to warn in case of disfunctioning. Patients can fill their medication cassette themselves. Furthermore they can learn how to program the CADD I-pump. This enables the patients to become independent of frequent medical intervention. A central catheter is necessary because otherwise a flebitis will rapidly develop.

Three very instable asthma patients although each using 40-60 mg prednisolon/day used the CADD I- pump. Patients filled in an asthma score (range 0-18) and recorded their PEFR on 8.00 and 20.00 hour before and during continuous ambulant theophylline infusion. FEV1, VC, R_{total}, RV, MEF50 were recorded before as well as during pump therapy and are expressed as percentage of the predicted EGKS-values. GAW is measured also before and during pump therapy and is expressed in L/kPa.S. The results are exoressed in Table 1.

Table 1	Asthmascore	PEFR 08.00 u	PEFR 20.00 u	R tot	GAW	RV	VC	FEV1	MEF50
Patient A									
Before pumptherapy	12.0±1.7	146.8±41.8	135.0±21.2	115.3	0.99	140.5	101.9	102	91.2
2 weeks pumpther	0.2±0.4	294.2±25.0	314.2±19.6	105.7	1.4	67.3	102.7	103	98.7
Patient B									
Before pumptherapy	12.0±3.2	105.0± 7.0	112.0±17.6	211.6	0.58	103	43.7	34.7	26.5
6 weeks pumpther	8.0±2.5	231.6±53.6	260.0±27.0	151.9	0.6	132.3	87.9	57.4	25.6
Patient C									
Before pumptherapy	9.5±1.6	69.0±11.0	130.0± 5.0	234.7	0.52	144.6	98.5	55.6	23
2 weeks pumpther	7.4±1.7	81.0±18.0	160.0±16.0	132.4	0.92	118.6	111.	82.2	39.3

In conclusion one can say that continuous intravenous therapy with theophylline can be a resource for brittle asthmatic patients. Therapy is given ambulatory and is an acceptable form of treatment.

RUSSIA

INTENSIVE CARE OF ACUTE ASTHMA AT HOME.

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The eim of the work was to study the emergency medical care of acute asthma at home in patients with unstable psychoemotional status. Ninety-five patients were being observed for 2 to 5 years; 80% of them were women aged from 17 to 67. The avereage duration of the disease was 16 years. Over 93% of patients regularly used β_2-adrenoreceptor agonists in inhelations mostly and 59% petients received corticoateroids. In 83% of cases the changes were reveeled in psychoemotional status in the form of astheno-hypochondria (36%), hysteria (20%), phobia (17%) etc. Patients called for the emergency medical care due to acute eathma from 6 to 12 times a year. The ambulence with a team including a pulmonologist, a specialist on intensive care, and a doctor's assistant visited them at home. The urgent diagnostics included the blood gas and theophylline concentration control when necessary. The infusion, pharmacological and psychic therapies were conducted. The treatment wes effective in 66% of cases including 26% of patients refusing hospitalization.

Thus,intensive care of acute asthma at home reduces the frequency of recurrences and the necessity in hospitalization.

UNITED KINGDOM

HOME NEBULISER THERAPY DURING ACUTE EXACERBATIONS CHRONIC AIRFLOW LIMITATION: BIOCHEMICAL AND CARDIAC EFFECTS.

M.J. Walshaw, R. Lim, C.C. Evans and C.R.K. Hind
Department of Medicine, Royal Liverpool Hospital, University of Liverpool, Liverpool L7 8XP, United Kingdom.

A recent study of deaths in asthmatic patients using home nebulisers reported that 16-30% delayed seeking help because of over-reliance on high-dose $beta_2$-agonists (Brit Med J 1987;294:477-80). The exact mechanism of death in such cases is unknown, but could be due either to fatal arrhythmias induced by myocardial hypoxia, or to arrhythmias secondary to $beta_2$-agonist-induced hypokalaemia. To investigate this further, we prospectively studied the effect of such treatment on plasma potassium levels and cardiac rhythm in 20 patients admitted to hospital with an acute exacerbation of their revesible chronic airflow limitation (11 male, 9 female; mean age 67, range 29-81 years). A continuous 24-hour ECG was performed on the day of admission (Oxford Medilog System), during which the patient received nebulised salbutamol or terbutaline (5mg, qds) via an air-driven home nebuliser (Bard). Plasma potassium levels were measured immediately before and 45 minutes after the first nebulisation. We found potentially life-threatening arrhythmias (ventricular tachycardia or couplets, or frequent ectopics) in 13 patients (65%). However there was no significant increase in these arrhythmias in the hour following administration of nebulised $beta_2$-agonist despite a significant fall in plasma potassium level during this period (mean change -0.30 mmol/l, SD 0.41, $p<0.005$). In contrast the hourly frequency of supraventricular ectopics was significantly higher immediately following the nebulisation (postnebulisation mean 104.2, SD 162.2: prenebulisation mean 84.9, SD 139.4) ($p<0.01$); these changes however are considered to be benign and do not herald life-threatening arrhythmias. These results suggest that providing standard doses of $beta_2$-agonists are given no more than six-hourly during the acute illness, the use of air-driven home nebulisers does not seem to be associated with an increased risk of potentially life-threatening arrhythmias.

LONG-TERM OXYGEN

YUGOSLAVIA

FOLLOW UP OF PATIENTS ON LONG TERM HOME OXYGEN THERAPY

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The authors unexpectedly visited 60 patients (42 males aged 64±4,6 yrs and 18 females aged 63,4±9 yrs), who were on the long term home oxygen therapy (LTOT) from 0,5 to 4 years. All patients had oxygen concentrators. During the visit, "the Chronic Respiratory Disease Index Questionnaire" on the quality of life was used (Guyatt and all 1986), the clinical examination of the patients was done and the oxygen saturation of the arterial blood (SaO_2) was measured by pulse oxymeter (OHMEDA) during air breathing and during oxygen inhalation. The functioning of the oxygen sources was also controlled by oxygen monitor (OHIO).

38 (63%) patients were using oxygen when visited unannounced. 45 (75%) of patients used the oxygen concentrator more than 15 hours a day (from 1-2 l/min). 7 (11%) patients had clinical signs of right heart failure and less than one third of them had mild, mostly peripheral cyanosis. The clinical status was better in patients, who hadn't been in the final stage of the chronic obstructive pulmonary disease (COPD) when starting the LTOT. SaO_2 during air breathing was 83,8±7,9% and during oxygen inhalation 91,6±8%. 4 (6,5%) concentrators were found damaged.

After the introduction of LTOT the number of hospitalizations was diminished for 74% and the duration (in days) decreased for 80% as well. In general, the patients had also less acute exacerbations of COPD.

According to the patients' (subjective) evaluation two thirds of them found their quality of life improved, but while measured by the questionnaire it was scarcely satisfactory only in the half of them. Analyzing the data obtained by the questionnaire, the degree of dyspnoe, physical activity and emotional function was correlated to the determined quality of life.

During the conversation 10 (16%) patients admitted the suicidal thoughts when starting the LTOT, while most of them complained of the continuous feeling of loneliness. The obligatory visits of medical personnel were found irregular and too rare in one third of the patients.

Though the results of the LTOT are generally successful, the medical and technical control of the LTOT should be improved in the future and the appropriate medical and social rehabilitation programmes for these patients should be planned and organised as well.

UNITED KINGDOM

LONG TERM HOME OXYGEN THERAPY (LTOT) USING OXYGEN CONCENTRATORS: FACTORS INFLUENCING PATIENT COMPLIANCE.

M.J. Walshaw, R. Lim, C.C. Evans and C.R.K. Hind
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Recent work on the use of LTOT given by home oxygen concentrators has shown that many patients fail to use their machines correctly (Brit Med J 1988;297:1030-3). In an attempt to discover which patients are more likely to be compliant with treatment, we studied 61 of the 65 adult patients who were using oxygen concentrators for LTOT in one city of the UK. Based on the concentrator meter readings, we found that 28 patients (group I) ran their concentrators for the recommended minimum time of at least 15 hours per day (mean 19.4, SD 3.2), and the remaining 33 patients (group II) for less than this (mean 10.7, SD 2.8). This was despite the fact that there was no significant difference in the amount of oxygen prescribed by the doctor between the two groups (group I mean 14.2 hours, SD 4.3; group II mean 12.5 hours, SD 4.1). Patients in group I were more hypoxic than patients in group II both on breathing air (group I mean PaO_2 6.73 kPa, SD 1.21; group II mean PaO_2 7.97 kPa, SD 1.65) ($p<0.001$) and also on supplementary oxygen treatment (group I mean PaO_2 8.54 kPa, SD 1.92; group II mean PaO_2 9.96 kPa, SD 1.88) ($p<0.001$), suggesting that patients in group I had more severe respiratory disease. However, there was no significant difference in respiratory symptoms, smoking habits, spirometry, or haematocrit between the two groups. In terms of the patients' estimation of their concentrator usage, patients in group I were greater (mean 18.0 hours, SD 3.7) than those in group II (mean 14.8 hours, SD 3.4) ($p<0.001$), a result confirmed by the actual concentrator meter readings (detailed above). Finally, we found that patients in group I were more likely to have been assessed by a respiratory physician in hospital (21 out of 28 cases) than patients in group II (13 out of 33 cases), suggesting that they may have had better advice regarding concentrator usage. These results suggest that patients with more severe gas exchange problems are more likely to use their concentrators appropriately. This may reflect either the patients' perception of benefit from LTOT, or better instruction from their specialist.

SPAIN

ONE YEAR FOLLOW-UP FOR PATIENTS WITH HYPOXIC CHRONIC AIRWAYS OBSTRUCTION, RANDOMISED BETWEEN ALMITRINE BISMESYLATE AND PLACEBO (VIMS STUDY)

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Almitrine bismesylate (Abm) a specific chemoreceptor agonist has been shown to be useful in the treatment of chronic airways obstruction with hypoxemia although caution has been raised about peripheral nerves disturbances after chronic administration.

2 centres in Spain participated to a double-blind placebo-controlled trial using a constant 50 mg bid dosage of the drug over one year. 48 patients (M=45; F=3), mean aged 60.5 ± 7.1 (sd) yr with advanced airways obstruction FEV_1 = 855 ± 276 ml, PaO_2 = 55.9 ± 5.8 mmHg, $PaCO_2$ = 52.0 ± 6.0 mmHg entered the study after a 3 week stabilisation period. They were randomly allocated to receive, in addition to conventional therapy (including oxygen), Abm (group A, n = 24) or identical placebo tablets (group P, n = 24), twice a day with quarterly examinations including clinical and neurological examination, arterial blood gas, laboratory measurements and pulmonary function tests.

The 2 groups were comparable on entry. 43 patients completed one year of treatment, 2 patients died (1 A, 1 P), 1 withdrew voluntary (1 P) and 2 for minor side effects (1 A, 1 P). Group P was stable except for a worsening of FEV_1 and the need for oxygen treatment in 2 cases. Group A demonstrated a significant PaO_2 improvement vs baseline: +6.7 mmHg after one year with a plateau after 3 months ($p<0.05$ in comparison with group P). $PaCO_2$ decreased (-3.7 mmHg, $p<0.05$) without significant changes in external ventilation. As a consequence, red blood cell count was significantly reduced from 5.56 to 5.38 10^{12}/L ($p<0.01$). Interestingly, FEV_1 was slightly improved + 22 ml ($p<0.01$) over one year.

A significant decrease of weight of 9% was observed after Abm, in one centre where no nutritionel supplements were given; on the contrary, no significant weigth loss was observed in the second centre where a high lipid and protein diet was encouraged. Otherwise, clinical and neurological examination was unchanged.

In conclusion, this placebo-controlled study demonstrated that Abm 50 mg bid improved major indices of severity in patients with chronic airways obstruction.

SPAIN

HOME CARE IN PULMONARY PATIENTS WITH DOMICILIARY OXYGEN THERAPY. ONE YEAR HOSPITALIZATION RATE AND MORTALITY.

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For one year we studied the incidence of home care (HC) on survival and days of hospitalization in a group (HCG) of 17 chronic respiratory patients who were undergoing continuous oxygen therapy. The HC team was made up of two nurses (NS) and a trained general practitioner (GP). The NS visited the patients at least each 15 days, and as often as necessary, depending on the judgement of the GP. The GP routinely visited the patients once a month, moreover, depending on the subjects' situation and as requiried by the NS. The latter (NS) apart from checking the emotional situation of the patients and their compliance with treatment, looked for so-called "signs and symptoms of alarm" by means of programmed anamnesis and physical examination. A pneumologist (PN), who checked the patients each 30 days in a specialized center, or upon request of the GP, completed the procedure. Thus the patients were seen at least four times per month. One group of 14 patients with similar characteristics, and who received regular medical atention (which, in our country, is provided by both a GP without specific trainning and a PN) served as a control group (CG). There were no significant differences ("t"-test) in age (HCG = 65±9 yrs; CG = 65±7 yrs) the % of theoretical FEV_1 (HCG = 26±1 %; CG = 26±9 %), $PaCO_2$ (HCG= 57±9 mmHg; CG= 55±1 mmHg), PaO_2 (HCG= 47±7 mmHg; CG= 50±6 mmHG) and hematocrit (HCG= 53±4 %; CG= 53±5 %) between the two groups at the beginning of the protocol. At the end of one yesr, ther were fewer days of hospitalization in the HCG (Chi-square= 136.17; $p<0.001$) (HCG= 64 days; CG= 234 days), cost of hospitalization was lower (HCG= 12,800 $; CG= 46,800 $) and also lower (Chi-square= 3.87; $p<0.05$) was the number of patients hospitalized (HCG= 5 patients; CG= 10 patients). Two of the HCG (12%) and three of the CG (21%) patients died. We conclude that, in our group, the specific HC improved survival, reduced hospitalization, and lowered costs, with resulting benefits for the pstients and savings for Social Segurity.

LONG-TERM OXYGEN

UNITED KINGDOM

HOME OXIMETRY IN PATIENTS USING OXYGEN CONCENTRATORS FOR LONG-TERM OXYGEN THERAPY (LTOT).

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The guidelines for LTOT suggest that the daytime PaO_2 while using oxygen delivered by a concentrator should be >8kPa. A recent review of such patients in the city of Liverpool showed that only 42% achieved this PaO_2 (Thorax 1988;43:860P). Despite the improved daytime PaO_2 and correct daily use of the oxygen concentrator (as assessed by meter readings), some patients were found to still have an elevated haematocrit after more than 6 months LTOT. In order to investigate this further we undertook domiciliary oximetry in these patients with three aims in mind. Firstly, to assess the adequacy of overnight oxygenation, secondly to exclude the presence of obstructive sleep apnoea which might prevent satisfactory oxygenation; and finally to determine the acceptability of domiciliary oximetry to patients. We studied 6 patients (2 male, 4 female, 57-66 years) who had used LTOT for between 3 and 18 months, and whose daytime PaO_2 was >8kPa whilst taking oxygen. An Ohmeda Biox 3700 pulse oximeter was used to monitor the patients in their own home. All subjects slept in their normal position using oxygen delivered via nasal cannulae at 2l/min. We found the technique to be simple and was readily tolerated by the patients, and that sleep quality was reported as representative of a usual nights sleep. Awake oxygen saturation (SaO_2) was >90% in all patients. However, only three patients sustained an SaO_2 >90% for more than 90% of the monitoring time. A further two patients spent <50% of the recording time with an SaO_2 >90%. Overall, the minimum overnight SaO_2 ranged from 72-87%, and no patient had evidence of obstructive sleep apnoea. In the light of these findings an increase in oxygen flow rate was recommended for the two patients with the worst desaturation overnight. In summary, we found that in those patients not apparently benefitting from LTOT despite adequate daytime arterial blood gas tensions, domiciliary oximetry was a simple and well tolerated technique which has helped in their management.

POLAND

LONG-TERM OXYGEN THERAPY IN POLAND

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Long-term oxygen therapy (LTOT) based on oxygen concentrators(OC) was introduced in Poland in 1986. The LTOT system belongs to the national health care, to its part responsible for care for lung diseases and is run by pneumonologists. OC BX-5000 assembled in Poland are bought by local centres of lung care and given to patients free of charge. System is centralized. Qualification criteria and follow-up scheme developed by Institute of Tb and Lung Diseases (ITbLD) are observed throughout the country. ITbLD is responsible for training of personnel working in provincial LTOT centres. Up to now in 20 out of 50 provinces of Poland such centres equipped with around 600 OC were opened. To the main LTOT centre, attached to ITbLD in Warsaw, during 2 years of work 133 pts were referred, 89 of whom were qualified for LTOT. The remaining pts improved during probatory period and fulfilled no more the inclusion criteria. Among 89 pts treated were 57 males and 32 females, mean age 56-12 y. Majority (73) pts suffered from COPD, 5 had IPF, 5 bronchetases, 6 had other lung diseases. Physiological variables of qualified pts at the time of qualification were as follows: (mean values) PaO2=56-7mmHg, PaCO2=47-10mmHg, FEV1=0.91-0.51, PCV=50-6%, pulmonary arterial pressures (PAP)=31-13mmHg. ECG signs of RVH were found in 68 pts, radiological signs of pulmonary hypertension were found in 67 pts. From 89 pts treated with LTOT 26 pts died. 32 COPD pts were reexamined after one year of LTOT. In those pts, during one year of treatment, mean PaO2 fell by 2mmHg, PaCO2 increased by 1mmHg, FEV1 fell by 130ml, PCV fell by 1% and PAO by 1 mmHg. Mean O2 breathing time was 14,5 H/day

SPAIN

DETERMINATION OF THE CLINICAL STEADY STATE IN CANDIDATES FOR LONG-TERM OXYGEN THERAPY (LTO)

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A probatory period on an optimal therapeutic regimen is necessary before prescription of LTO. Our study tried to determine the time necessary to ascertain the stability of hypoxemia and consequently the indication for LTO.

Materials and methods.- In a 12 month period, 50 patients with severe COPD were included at discharge of a hospital care for an acute respiratory failure. Those patients had never been previously treated with LTO. Patients older than 75 yr of age or with a history of asthma attacks were excluded. At the end of hospital care (TO) the patients were distributed in 2 groups depending of their PaO2: Group A: PaO2 between 50 and 55 mmHg, Group B: PaO2 between 55 and 60 mmHg. During a probatory period of 3 month, patients received at home full drug therapy, were asked to continue their daily chest physiotherapy exercices and were checked monthly at the out-patient department (T1, T2, T3) and ABG were measured every time. Mean values of age, spirometry, pulmonary volumes, hematocrit and hemoglobin were not significantly different in the two groups at TO. The arterial blood gas at TO according the group were: Group A (n=23) PaO2 53 6±1.4; PaCO2 53.4±5.5; pH 7.38 ± 0.03. Group B (n=27) PaO2 57.9 ± 1.5; PaCO2 49.6 ± 7.5; pH 7.33 ± 0.03.

Results: Only 37 patients could be followed regulary to T3. In group A (23 patients), 6 have been excluded for repeated exacerbations. In group B (27 patients), 7 have been excluded (1 for repeated exacerbations, 5 did not accept the medical regimen, 1 lost to follow-up). In group A, the mean ± 1 SD of PaO2 improved from 53.6 ± 1.4 at TO to 58.4 ± 5.1 at T3. In 4 patients, the PaO2 persist inferior to 55 mmHg at T3 (2 were not identifiable at T1 and T2). In group B, the mean ± 1 SD of PaO2 improved from 57.9±1.5 at TO to 63.7±7.8 at T3. In 3 patients, the PaO2 was inferior to 55 mmHg at T3 (none was identifiable at T1 and T2).

Conclusions: 1) The previous follow-up period before prescription of LTO after hospital discharge is necessary and may preferably be of 3 month. 2) The initial level of PaO2 does not allow prediction of its evolution from TO to T3. 3) Age, spirometry, pulmonary volumes, hemoglobin and hematocrit cannot be considered as predictive at TO. (SEPAR-OXIFAR 1986 Grant)

SPAIN

CONTINOUS DOMICILIARY OXYGEN THERAPHY; REAL VERSUS REPORTED COMPLIANCE IN A GROUP OF PULMONARY PATIENS. E. Servera, J. Marín*, P. Vergara, L. Simó*, M.E. Pérez*. University School Departament of Physiotherapy and * Departament of Medicine (Pneumology Unit). Valencia, Spain.

In a recent paper, P. Sadoul (1) emphasizes the necessity of patients with domiciliary oxygen therapy (DOT) having not less than 18 hours/day of treatment; in medical practice many patients claim that they comply with this norm. In the phase prior to a home-care protocol we studied a group of pulmonary patients to determine whether the real time of oxygen therapy corresponded to that which they claimed to have.

Eleven patients with Chronic Airflow Obstruction (CAO) undergoing DOT for at least a year received, for two months, a De Vilbis Devo 44 concentrator which a hidden hour meter. All were informed they should have as many hours as possible of DOT, and always more than 15 hours/day.

The mean age of patients was 63±5 yrs and all suffered from serious bronchial obstruction (FEV1=24±8 % of the theoretical value), marked hypoxemia (PaO2= 51±4 mmHg) plus hypercapnia (PaCO2= 54±10 mmHg). Hematocrit was high (53±3%) In spite of all patients insisting on having had at least 15 hours/day DOT for two months, checking the hour meters revealed that only one subject managed an average of 15 hours/day; five fell between 12 and 15 hours/day, two showed 10 hours/day, one only 6 hours/day, and another two but 3 hours/day: The mean hours of DOT in this group was merely 10±4, far below that desired.

We conclude that, at least in these patients, the real time of DOT in hours/day did not correspond to what was claimed, and suspect some failures in the home therapy of CAO patients could be due to poor compliance of which the pneumologist is unaware.

(1) Rev. Mal. Resp. 1988, 5: 363-372

LONG-TERM OXYGEN

BELGIUM

THE IMPORTANCE OF BEING EARNEST :
FO2 CONTROL OF O2-CONCENTRATORS.

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To be efficient, Long Term Oxygen Therapy (LTOT) needs a good compliance of the patient and reliable equipment. Oxygen concentrators (O2 conc) are largely used but the delivery of an adequate % of oxygen (FO2) depends upon the O2 flow rate and the perfect working of the device.
Material and Methods. - Since 1984, we purchased 56 O2 conc: 49 Mini O2, 3 Delox 1, 2 Sriox PDQ. - LTOT was prescribed for at least 15 hours a day at a O2 flow rate of 2L/min. - Maintenance and repair are ensured by our lung function lab. and in some cases by the manufacturers. - FO2 controls are performed with a mass spectrometer or a portable analyser (TI-Vaase industries S.A.) - Home visits are performed on a regular basis by a technician or a nurse.
Results. - 125 patients were included in our LTOT program for a mean duration of 11 ± 10 months. - FO2 of the O2 conc was controlled 491 times at home and 116 times in the lab., at an interval of about 1000 hours of function.
- An insufficient FO2 was detected in 49 cases during a systematic control but only in 7 cases by the patient himself.
- The major problem with the Mini O2 was a failure of the sieve-beds after a period of time ranging from 300 to 14000 hours of function. Only 14 out of the 49 devices had no major problems; 27 had 1 failure; 8 had 2 failures.
- We had no major problems with the Delox 1 after a mean use of 17000 hours but repetitive and quite expensive break downs with Sriox PDQ.
- There was no progressive decline of FO2 and all failures were unpredictable.
Conclusions :
(1) A dramatic and unpredictable decrease of FO2 is frequent.
(2) In the majority of cases, the patient is not aware of the failure of his device.
(3) In order to provide an efficient LTOT, we need frequent home controls or the development of a monitoring of O2 delivery.

HOME CARE AND REHABILITATION

FEDERAL REPUBLIC OF GERMANY

NONINVASIVE INTERMITTENT VENTILATION-INDICATION BY RESPIRATORY MUSCLE FUNCTION G.Laier-Groeneveld, C.-P. Criée, U. Hüttemann. Krankenhaus an der Lieth, Pneumologische Klinik, D 3406 Bovenden-Lenglern, FRG

The respiratory muscles have contractile properties similar to other skeletal muscles and therefore fatigue may develop and contribute to hypercapnic ventilatory failure. One benefit may be the total resting of the respiratory muscles for sufficient time for fatigue to resolve by noninvasive intermittent ventilation via a nasal mask (NIV).

We studied 9 patients (6 patients with neuromuscular disorders and scoliosis, 3 patients with obstructive lung disease) with chronic muscular weakness (PImax < 60 cm H_2O) and chronic ventilatory failure during NIV

Within four weeks of NIV, spontaneous tidal volume increased from 0,43 ± 0,2 to 0,52 ± 0,3 l ($p < 0,01$). Po.1, an index of inspiratory pressure generation, was increased in all patients (Po.1 = 4,8 ± 1,3 cm H_2O) before therapy and did not change significantly. Maximum inspiratory pressures, PImax and Po.1max, increased from 44 ± 11 cm H_2O to 58 ± 17 cm H_2O and 19,3 ± 3,9 to 25 ± 77, respectively ($p < 0,05$, $P < 0,02$). The actual demand on the inspiratory muscles was assessed by the Po.1/Po.1max-ratio. It decreased from 25± 5 to 19± 4 %. The pressure-time-index (calculated by Po.1/Po.1max · T_I/T_{TOT}), decreased from 0,09± 0,02 to 0,07± 0,02 ($p < 0,001$), indicating a reduced risk in developing muscle fatigue. PO_2 increased from 56± 5 to 68± 2 Torr ($p < 0,001$) and PCO_2 decreased from 51± 9 to 47± 5 Torr (n.s.). We conclude that NIV is sufficient to reduce the demand on inspiratory muscles by increasing muscular strength. Therefore, NIV is indicated in patients exhibiting inpaired muscle strength (PImax < 60 cm H_2O) and/or increased demand on inspiratory muscles during quiet breathing (Po.1/Po.1max > 15 %).

ITALY

SHORT AND MID-TERM EFFECTS OF HOME MECHANICAL VENTILATION IN HYPOXEMIC HYPERCAPNIC REPIRATORY FAILURE.

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The aim of our study was to check the results of home mechanical ventilation (HMV) by tracheostomy. Methods: We studied two groups of pts with chronic hypoxemic-hypercapnic respiratory failure. The former group included 7 COPD pts (mean age 63±6) and the latter included 3 restrictive lung disease (RLD) pts (1 post-polio, 2 kyphoscoliosis, mean age 59±7). All pts were tracheostomized and ventilated by volumetric device (Monnal-D) for 10±2 hs/day. Two pts had been treated before by long term oxygen-therapy (18±3 hs/day) and other four by IPPV (tight fitting mask). After a short period in ICU and an other period (20±5 days) in Rehabilitation Center RICU the pts were discharged with home mechanical ventilation. After discharge we checked these parameters: pH, PaO2, PaCO2 at rest every 20 days, and Htc every 4 months.
Results:

COPD

	Before	Latest data
ph	7.34±0.02	7.39±0.01(*)
PaO2	41±8.2	58±2.31(§)
PaCO2	72±12.1	45±2.5(*)
Htc	52±6.9	41±6.3(*)

RLD

	Before	Latest data
ph	7.37±0.03	7.40±0.04
PaO2	43±10.0	69±5.2
PaCO2	66±12.3	38±5.0
Htc	44±1.0	37±3.0

t-Student's test: (*) $p<0,005$ - (§) $p<0,001$
Months of HMV: COPD = 6.8 (1-26) ; RLD = 7.3 (4-9)
Hospitalization(days/year,before/after):
COPD = 31±8/5±2 ; RLD = 38±2/6±1
Conclusions: in COPD patients 1) there was a significant improvement of blood gases; this improvement appeared in the 1st month yet without appreciable variation in the follow-up period (data not shown) 2) all the pts had a sharp fall in hospitalization (after HMV onset they were mainly admitted for a planned follow-up programme). In RLD patients the same trend as in COPD can be observed, but data are not significantly different due to the small number of patients.

NUTRITION

NETHERLANDS

WEIGHT LOSS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD): "DECREASED DIETARY INTAKE OR INCREASED ENERGY EXPENDITURE?"

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Weight loss is frequently encountered in the course of severe COPD. The relative importance of anorexia and abnormal energy metabolism in the development of weight loss was investigated in 70 COPD patients (IVC%pred. 69 ± 20; FEV_1%pred. 37 ± 17), admitted to a pulmonary rehabilitation centre. Resting energy expenditure (REE: ventilated hood) and dietary intake (cross check dietary history) were expressed per kg body weight (BW) and lean body mass (LBM: bioelectrical impedance), and as a percentage of predicted REE according to the Harris & Benedict equation (H&B). Mean values, stratified for energy expenditure and weight loss, are displayed in the table.

		REE > 24 kcal/kg		REE ≤ 24kcal/kg		
weight loss		yes [1]	no [2]	yes [3]	no [4]	[1-2] / [3-4]
			[1-2]			
REE	kcal	1524	1608 *	1396	1470	*
REE/H&B	%	125	121 *	103	103	*
REE/BW	kcal/kg	28.8	26.0 *	21.5	21.0	***
REE/LBM	kcal/kg	36.7	35.4	31.2	31.6	***
INTAKE	kcal	1837	2232 *	1632	1882	
INTAKE/H&B	%	152	167	120	134	
INTAKE/REE	%	121	139 *	116	130	
INTAKE/BW	kcal/kg	34.7	36.1	24.5	27.5	****
INTAKE/LBM	kcal/kg	44.4	48.9	35.5	41.	***

* p<0.05, *** p<0.005, **** p<0.001

36 (51%) patients recalled a history of non voluntary weight loss, accompanied in 69%[1] by an increased energy expenditure. Dietary intake was significantly higher in the hypermetabolic group (n=36)[(1-2)], suggesting that hypermetabolism may be compensated by increasing dietary intake. In both sub-groups [(1/2+3/4)] patients suffering from weight loss tended to eat less than patients with stable body weight.

It is concluded that both energy expenditure and dietary intake should be considered in the evaluation of weight loss in COPD patients.

NETHERLANDS

DOES NUTRITIONAL STATUS INFLUENCE EXERCISE PERFORMANCE IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)?

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Many patients with chronic obstructive pulmonary disease are severely disabled by breathlessness and exercise impairment. Recently several studies indicated an association between impaired nutritional status and COPD. We studied a possible relationship between exercise performance and some commonly used parameters of nutritional status in 83 COPD patients, admitted to a pulmonary rehabilitation centre.

All patients were in stable clinical condition. Mean FEV_1 was less than 50% of predicted. Criteria for exercise testing were a P_aO_2 > 7.3 kPa and no severe locomotory, cardiovascular, neurological or endocrine disorders. Exercise performance was evaluated with a 12 minute walking test. Percentage of ideal body weight (PIW), creatinine height index (CHI) as an indicator of muscle mass, and the visceral proteins albumin (ALB), prealbumin (PALB) and transferrin (TRF) were assessed as parameters for nutritional status.

Mean walking distance (WD) amounted to 686 ± 254 metres. WD correlated significantly with ALB ($r = 0.44$; $p < 0.001$) and CHI ($r = 0.35$; $p < 0.005$, but not with PIW ($r = 0.19$, NS), TRF ($r = 0.09$, NS) and PALB ($r = 0.10$, NS). Furthermore the parameters were stratified for walking distance into 3 groups (cut-off points: WD ± 1.sd). Mean CHI was very low (59 ± 19%) in the low performance group. Stepwise regression analysis revealed that ALB significantly explained part of the variance in WD independent from inclusion of any of the lung function parameters in the model. The best fitting equation ($R^2 = 0.33$) included ALB, P_aO_2 and age.

The finding that both CHI and ALB were associated with WD suggests that not only loss of muscle mass but also metabolic factors contribute to a limited exercise performance in COPD patients.

Right-Side Thoracic Pain in a Young Woman

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Alessandra Fontana MD, Stefano Nazari MD, Paolo Cremaschi MD,
and Caterina Nascimbene MD

A 17-y-old woman was admitted to our institution complaining of right-side thoracic pain. The woman did not have a family history of pulmonary disease, but she had received treatment 1 year previously for a right pleural effusion of unknown etiology.

Upon admission, the woman was apyretic, with no signs of systemic disease. Chest auscultation revealed dullness (with an absence of vesicular breath sounds) in the right lung base. No hemoptysis or cough were present. Anteroposterior (AP) and lateral chest radiographs obtained on admission are shown in Figure 1.

Questions

1. **Findings:** What do these radiographs reveal?
2. **Further Tests:** What other diagnostic tests are indicated?

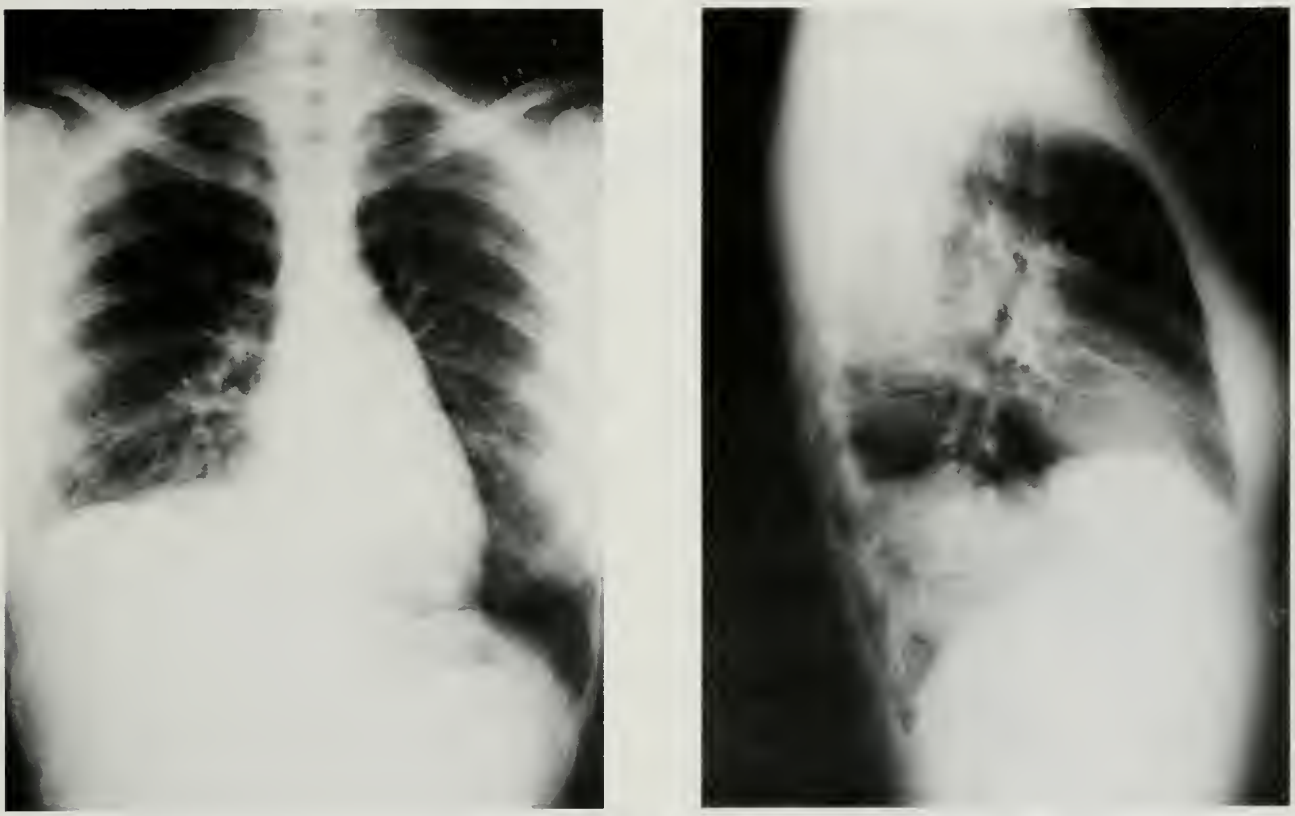

Fig. 1. Anteroposterior (1a) and lateral (1b) chest radiographs of 17-y-old woman complaining of right-side thoracic pain.

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Answers and Discussion on next page

Answers

Findings: The AP chest radiograph shows a pleural effusion, no enlargement of the mediastinum or mediastinal structures, and no shift in the midline. The lateral chest radiograph reveals the presence of both pleural fluid and a pleural-based mass.

Further Tests: With pleural fluid and mass visible in the simple chest radiographs, we did not believe further radiologic testing was indicated. A pleural fluid sample for cytologic examination and a pleural-mass-tissue sample for histologic examination were required for diagnosis. We chose to perform thoracoscopy for this purpose instead of the less invasive needle biopsy, both to obtain a larger tissue sample for examination and to obtain more details concerning the extent of disease progression.

Discussion

Diagnosis: Thoracoscopy was carried out, and multiple masses and nodules of variable dimensions were found involving both visceral and parietal pleura. Biopsy showed a few fusate cells in a rich collagen matrix, diffusely hyalinized; the cellularity was very scarce wherever the hyalinized collagen matrix was predominant. The cytologic features of the fibroblasts of the tumor were regular and uniform; in particular, no mitosis could be seen (Fig. 2). Histology showed patterns of a benign fibrous tumor.

Benign fibrous tumors of the pleura are usually referred to as benign localized fibrous mesotheliomas, thus pointing out their origin from the mesothelial cells and their localized growth. However, some authors have suggested that they originate in submesothelial connective tissue, and others consider these tumors to be the end result of a low-grade inflammatory process with organization of the exudate.[1,2] These localized tumors are usually in the form of sessile or pedunculated masses of the parietal or visceral pleura, and occur with equal frequency in male and female subjects. Occasionally, they may reach great dimensions; however, they are usually well encapsulated, do not metastasize, and are not likely to recur after surgical removal.

In our case, it seemed appropriate to plan a thoracotomy without delay to validate (with a larger biopsy specimen) the suspected diagnosis, to assess the extent of intrathoracic tumor spread, and to perform resection as indicated. During thoracotomy, widespread diffusion of the mass throughout the whole hemithorax was confirmed; the parietal pleura was diffusely involved, mainly in the area of the right costophrenic angle. A few nodules were also found on the visceral pleura of the right lower lobe and on the diaphragmatic and mediastinal pleura.

Treatment: In planning the treatment, we decided to completely remove the parietal pleura, which was the most diffusely involved, with the aim of preventing possible recurrence at that site. The numerous masses on the diaphragmatic and mediastinal pleura and those on the visceral pleura of the lower lobe were each carefully removed.

The surgical sample consisted of many lobulated masses (Fig. 3) of variable size; the shape varied from

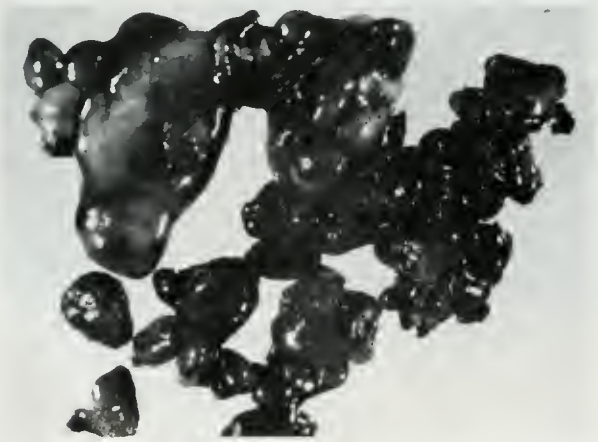

Fig. 2. Histologic appearance of thoracoscopic biopsy specimen. Normal fibrous tissue with low cellularity and rich, predominantly hyalinized collagen matrix; no mitosis; no malignant cells.

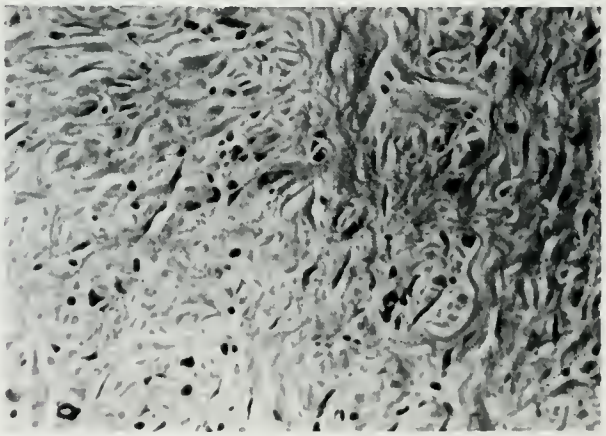

Fig. 3. A sample of the nodules removed (from the area of the right costophrenic angle) during thoracotomy.

plaques of different depth to huge pseudopolypoid masses. The surface was uniformly smooth and whitish-gray in color. No hemorrhage or necrosis was found on the cut surface, which was uniformly whitish and quite hard. Histology confirmed the previously described pattern of benign fibrous mesothelioma.

Two drains were positioned, and the thoracotomy was closed. The recovery of the patient was uneventful, and she is well 1 year after surgery.

Although localized forms of fibrous mesothelioma are often benign,[3] diffuse forms are usually malignant. We could find only two reports in the literature of diffuse, histologically benign, fibrous mesotheliomas.[2,4] With the exception of the case reported by Scattini,[4] in which the tumors recurred 12 years after resection, we were unable to find data concerning the recurrence rate of these tumors. However, due to the extremely diffuse pattern observed in this case, recurrence or subsequent growth of small lesions on the preserved visceral, mediastinal, or diaphragmatic pleura (overlooked during surgery) is at least possible. Future malignant changes, as seen in localized fibrous mesothelioma,[5] are also possible.

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Reducing the Health Consequences of Smoking: 25 Years of Progress. A Report of the Surgeon General (1989), prepared by the Department of Health and Human Services under the general editorship of the Office on Smoking and Health, Ronald M Davis MD, Director; Managing Editors—Susan Hawk EdM MS and Thomas E Novotny MD, Medical Epidemiologist, Office on Smoking and Health. Soft-cover, 687 pages. Available from the Superintendent of Documents, Government Printing Office, Washington DC 20402-9325. $23.00.

On January 11, 1964, the first Surgeon General's Report on Smoking and Health was released. This landmark report concluded that "Cigarette smoking is a health hazard of sufficient importance in the United States to warrant appropriate remedial action." Between 1964 and 1988, twenty reports on smoking and health have been released by the Surgeon General, each building upon the knowledge of its predecessors. The 1989 Surgeon General's Report reviews the progress against smoking that has occurred over the past 25 years (ie, advances in knowledge, changes in norms and behavior, and effects on the health of the American people).

The 25th Anniversary Surgeon General's Report on Smoking and Health was produced through the combined efforts of over 130 scientists from the fields of medicine, biological and social sciences, public health, and policy analysis. The report begins with a Foreword written by Robert E Windom MD, Assistant Secretary for Health, and James O Mason MD DrPH, Director of the Centers for Disease Control, Public Health Service, and with a Preface written by former Surgeon General C Everett Koop MD ScD.

Chapter 1 (Historical Perspective, Overview, and Conclusions) provides an excellent table that recaps the subject and highlights of each Surgeon General Report released from 1964 through 1988. An overview of the entire 1989 Report (with chapter conclusions) is also provided.

The 1964 Surgeon General's Report was based upon an analysis of more than 7,000 available scientific articles on smoking and health. It was concluded that cigarette smoking had a causal relationship with a specific disease only when the evidence was (1) consistent, (2) strong, (3) specific, (4) supportive of appropriate temporal relationships, and (5) coherent. The 1989 report, based upon an analysis of over 57,000 available documents on smoking and health, not only confirms that smoking has a causal relationship with many of the diseases that were suspect in 1964 but also confirms the causal relationship between smoking and many previously unsuspected disease entities or complication groups (unsuccessful pregnancies; increased infant mortality; peptic ulcer disease; cancer of the bladder, pancreas, kidney, and stomach; stroke; and uterine cancer). Chapter 2 (Advances in Knowledge of the Health Consequences of Smoking) summarizes the current knowledge of both the health consequences of tobacco smoking and the physiochemical nature of tobacco smoke.

Chapter 3 (Changes in Smoking-Attributable Mortality) examines the variations in the rates and sociodemographic distribution of smoking prevalence that have resulted in changing patterns of mortality in the U.S. during the past 25 years. Chapter 4 (Trends in Public Beliefs, Attitudes, and Opinions about Smoking) reviews changes that have occurred in public knowledge and sentiment regarding smoking since the 1964 Surgeon General's Report. The airline passengers survey conducted by the AARC in 1987 is cited in Chapter 4 as instrumental. Chapter 5 (Changes in Smoking Behavior and Knowledge about Determinants) goes a step beyond Chapter 4 and examines the actual changes in smoking behavior and prevalence that have occurred during the past 25 years. Specific population groups are compared on the basis of changing smoking behavior and prevalence. The psychologic and physiologic determinants of smoking behavior are also examined.

Chapters 6 and 7 (Smoking Prevention, Cessation, and Advocacy Activities and Smoking Control Policies, respectively) review the various educational approaches that have been employed to discourage would-be smokers from adopting the habit, the numerous cessation methods that have been used to assist smokers in breaking the habit, and the ever-increasing public policies that have been instituted to control smoking. Chapter 8 (Changes in the Smoking-and-Health Environment: Behavioral and Health Consequences) takes a step back and attempts to assess what the overall effect of the past 25 years of smoking prevention, cessation, advocacy, and control has been upon smoking behavior and health in the U.S.

The authors of the Foreword in the 1989 report state "We must look back on the last 25 years of change in order to look forward to our tasks for the future. . . . The Public Health Service is dedicated to continuing the legacy of the 1964 Report. We hope this 25th Anniversary Report will stimulate new commitment to action by public health officials, civic leaders, educators, scientists, and the public at large on the problem of tobacco use, especially among children, adolescents, and high-risk groups." In conclusion, I must say that this extensive report on smoking and health certainly does continue the legacy of the original Surgeon General's Report; it provides invaluable information and should be on the reference shelf of any respiratory care professional currently active in or contemplating becoming active in lung health promotion/smoking intervention.

Gary Peck BS RPFT RRT
Associate Editor
RESPIRATORY CARE
Dallas, Texas

"I never thought we could be hit...
I mean, I knew it was possible,
but what were the chances?"

RSV.
No one knows the path it will take.

The consequences may have a lasting effect. The short-term consequences of respiratory syncytial virus (RSV) infection can include nosocomial spread, mechanical ventilation, extended hospitalization—even death—and can threaten *all* hospitalized children under 4 years old. Respiratory viral infections may have a long-term effect on the respiratory tree in some patients, such as development of bronchopulmonary disease.[1]

Help reduce the toll taken by RSV. In clinical experience with over 50,000 patients, treatment of appropriate patients with Virazole® (ribavirin) Aerosol improved disease symptoms safely, speeding recovery[2-6] while significantly reducing viral shedding.[2,4] Virazole may reduce the need for supplemental oxygen and mechanical ventilation.[7] And Virazole has been shown to prevent development of RSV-specific IgE responses,[8] which may correlate with the release of histamine and clinical expression of obstructive reactive airway disease.[1]

Serious enough to hospitalize, serious enough to consider

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(ribavirin)
Lyophilized for aerosol administration

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Please see following page for full prescribing information.

STI-132

Serious enough to hospitalize, serious enough to consider

Virazole®
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PRESCRIBING INFORMATION

WARNING: RIBAVIRIN AEROSOL SHOULD NOT BE USED FOR INFANTS REQUIRING ASSISTED VENTILATION BECAUSE PRECIPITATION OF THE DRUG IN THE RESPIRATORY EQUIPMENT MAY INTERFERE WITH SAFE AND EFFECTIVE VENTILATION OF THE PATIENT. Conditions for safe use with a ventilator are still in development.

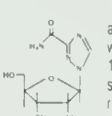
Deterioration of respiratory function has been associated with ribavirin use in infants, and in adults with chronic obstructive lung disease or asthma. Respiratory function should be carefully monitored during treatment. If initiation of ribavirin aerosol treatment appears to produce sudden deterioration of respiratory function, treatment should be stopped and re-instituted only with extreme caution and continuous monitoring.

Although ribavirin is not indicated in adults, the physician should be aware that it is teratogenic in animals (see CONTRAINDICATIONS).

DESCRIPTION:

Virazole® (ribavirin) Aerosol, an antiviral drug, is a sterile, lyophilized powder to be reconstituted for aerosol administration. Each 100 ml glass vial contains 6 grams of ribavirin, and when reconstituted to the recommended volume of 300 ml with sterile water for injection or sterile water for inhalation (no preservatives added), will contain 20 mg/ml ribavirin, pH approximately 5.5. Aerosolization is to be carried out in a SPAG-2 nebulizer only.

Ribavirin is 1-beta-D-ribofuranosyl-1,2,4-triazole-3-carboxamide, with the following structural formula:



Ribavirin, a synthetic nucleoside, is a stable, white, crystalline compound with a maximum solubility in water of 142 mg/ml at 25°C and with only a slight solubility in ethanol. The empirical formula is $C_8H_{12}N_4O_5$ and the molecular weight is 244.2 Daltons.

CLINICAL PHARMACOLOGY:

Antiviral effects:

Ribavirin has antiviral inhibitory activity *in vitro* against respiratory syncytial virus,[1] influenza virus, and herpes simplex virus. Ribavirin is also active against respiratory syncytial virus (RSV) in experimentally infected cotton rats.[2]

In cell cultures, the inhibitory activity of ribavirin for RSV is selective. The mechanism of action is unknown. Reversal of the *in vitro* antiviral activity by guanosine or xanthosine suggests ribavirin may act as an analogue of these cellular metabolites.

Immunologic effects:

Neutralizing antibody responses to RSV were decreased in ribavirin treated compared to placebo treated infants.[3] The clinical significance of this observation is unknown. In rats, ribavirin resulted in lymphoid atrophy of thymus, spleen, and lymph nodes. Humoral immunity was reduced in guinea pigs and ferrets. Cellular immunity was also mildly depressed in animal studies.

Microbiology:

Several clinical isolates of RSV were evaluated for ribavirin susceptibility by plaque reduction in tissue culture. Plaques were reduced 85-98% by 16 μg/ml, however, plaque reduction varies with the test system. The clinical significance of these data is unknown.

Pharmacokinetics:

Assay for ribavirin in human materials is by a radioimmunoassay which detects ribavirin and at least one metabolite.

Ribavirin administrered by aerosol is absorbed systemically. Four pediatric patients inhaling ribavirin aerosol administered by face mask for 2.5 hours each day for 3 days had plasma concentrations ranging from 0.44 to 1.55 μM, with a mean concentration of 0.76 μM. The plasma half-life was reported to be 9.5 hours. Three pediatric patients inhaling ribavirin aerosol administered by face mask or mist tent for 20 hours each day for 5 days had plasma concentrations ranging from 1.5 to 14.3 μM with a mean concentration of 6.8 μM.

It is likely that the concentration of ribavirin in respiratory tract secretions is much higher than plasma concentrations in view of the route of administration.

The bioavailability of ribavirin aerosol is unknown and may depend on the mode of aerosol delivery. After aerosol treatment, peak plasma concentrations are less than the concentration that reduced RSV plaque formation in tissue culture by 85 to 98%. After aerosol treatment, respiratory tract secretions are likely to contain ribavirin in concentrations many fold higher than those required to reduce plaque formation. However, RSV is an intracellular virus and serum concentrations may better reflect intracellular concentrations in the respiratory tract than respiratory secretion concentrations.

In man, rats, and rhesus monkeys, accumulation of ribavirin and/or metabolites in the red blood cells has been noted, plateauing in red cells in man in about 4 days and gradually declining with an apparent half-life of 40 days. The extent of accumulation of ribavirin following inhalation therapy is not well defined.

INDICATIONS AND USAGE:

Ribavirin aerosol is indicated in the treatment of carefully selected hospitalized infants and young children with severe lower respiratory tract infections due to respiratory syncytial virus (RSV). In two placebo-controlled trials in infants hospitalized with RSV lower respiratory tract infection, ribavirin aerosol treatment had a therapeutic effect, as judged by the reduction by treatment day 3 of severity of clinical manifestations of disease.[3,4] Virus titers in respiratory secretions were also significantly reduced with ribavirin in one of these studies.[4]

Only severe RSV lower respiratory tract infection is to be treated with ribavirin aerosol. The vast majority of infants and children with RSV infection have no lower respiratory tract disease or have disease that is mild, self-limited, and does not require hospitalization or antiviral treatment. Many children with mild lower respiratory tract involvement will require shorter hospitalization than would be required for a full course of ribavirin aerosol (3 to 7 days) and should not be treated with the drug. Thus the decision to treat with ribavirin aerosol should be based on the severity of the RSV infection.

The presence of an underlying condition such as prematurity or cardiopulmonary disease may increase the severity of the infection and its risk to the patient. High risk infants and young children with these underlying conditions may benefit from ribavirin treatment, although efficacy has been evaluated in only a small number of such patients.

Ribavirin aerosol treatment must be accompanied by and does not replace standard supportive respiratory and fluid management for infants and children with severe respiratory tract infection.

Diagnosis:

RSV infection should be documented by a rapid diagnostic method such as demonstration of viral antigen in respiratory tract secretions by immunofluorescence[3,4] or ELISA[5] before or during the first 24 hours of treatment. Ribavirin aerosol is indicated only for lower respiratory tract infection due to RSV. Treatment may be initiated while awaiting rapid diagnostic test results. However, treatment should not be continued without documentation of RSV infection.

CONTRAINDICATIONS:

Ribavirin is contraindicated in women or girls who are or may become pregnant during exposure to the drug. Ribavirin may cause fetal harm and respiratory syncytial virus infection is self-limited in this population. Ribavirin is not completely cleared from human blood even four weeks after administration. Although there are no pertinent human data, ribavirin has been found to be teratogenic and/or embryolethal in nearly all species in which it has been tested. Teratogenicity was evident after a single oral dose of 2.5 mg/kg in the hamster and after daily oral doses of 10 mg/kg in the rat. Malformations of skull, palate, eye, jaw skeleton, and gastrointestinal tract were noted in animal studies. Survival of fetuses and offspring was reduced. The drug causes embryolethality in the rabbit at daily oral dose levels as low as 1 mg/kg.

WARNINGS:

Ribavirin administered by aerosol produced cardiac lesions in mice and rats after 30 and 36 mg/kg, respectively, for 4 weeks, and after oral administration in monkeys at 120 and rats at 154 to 200 mg/kg for 1 to 6 months. Ribavirin aerosol administered to developing ferrets at 60 mg/kg for 10 or 30 days resulted in inflammatory and possible emphysematous changes in the lungs. Proliferative changes were seen at 131 mg/kg for 30 days. The significance of these findings to human administration is unknown.

Ribavirin lyophilized in 6 gram vials is intended for use as an aerosol only.

PRECAUTIONS:

General:

Patients with lower respiratory tract infection due to respiratory syncytial virus require optimum monitoring and attention to respiratory and fluid status.

Drug Interactions:

Interactions of ribavirin with other drugs such as digoxin, bronchodilators, other antiviral agents, antibiotics, or antimetabolites has not been evaluated. Interference by ribavirin with laboratory tests has not been evaluated.

Carcinogenesis, mutagenesis, impairment of fertility:

Ribavirin induces cell transformation in an *in vitro* mammalian system (Balb/C3T3 cell line). However, *in vivo* carcinogenicity studies are incomplete. Results thus far, though inconclusive, suggest that chronic feeding of ribavirin to rats at dose levels in the range of 16-60 mg/kg body weight can induce benign mammary, pancreatic, pituitary and adrenal tumors.

Ribavirin is mutagenic to mammalian (L5178Y) cells in culture. Results of microbial mutagenicity assays and a dominant lethal assay (mouse) were negative.

Ribavirin causes testicular lesions (tubular atrophy) in adult rats at oral dose levels as low as 16 mg/kg/day (lower doses not tested), but fertility of ribavirin-treated animals (male or female) has not been adequately investigated.

Pregnancy:

Teratogenic Effects: Pregnancy Category X. See "Contraindications" section.

Nursing Mothers: Use of ribavirin aerosol in nursing mothers is not indicated because RSV infection is self-limited in this population. Ribavirin is toxic to lactating animals and their offspring. It is not known whether the drug is excreted in human milk.

ADVERSE REACTIONS:

Approximately 200 patients have been treated with ribavirin aerosol in controlled or uncontrolled clinical studies.

Pulmonary function significantly deteriorated during ribavirin aerosol treatment in six of six adults with chronic obstructive lung disease and in four of six asthmatic adults. Dyspnea and chest soreness were also reported in the latter group. Minor abnormalities in pulmonary function were also seen in healthy adult volunteers.

Several serious adverse events occurred in severely ill infants with life-threatening underlying diseases, many of whom required assisted ventilation. The role of ribavirin aerosol in these events is indeterminate. The following events were associated with ribavirin use:

Pulmonary: Worsening of respiratory status, bacterial pneumonia, pneumothorax, apnea, and ventilator dependence.

Cardiovascular: Cardiac arrest, hypotension, and digitalis toxicity.

There were 7 deaths during or shortly after treatment with ribavirin aerosol. No death was attributed to ribavirin aerosol by the investigators.

Some subjects requiring assisted ventilation have experienced serious difficulties, which may jeopardize adequate ventilation and gas exchange. Precipitation of drug within the ventilatory apparatus, including the endotracheal tube, has resulted in increased positive end expiratory pressure and increased positive inspiratory pressure. Accumulation of fluid in tubing ("rain out") has also been noted.

Although anemia has not been reported with use of the aerosol, it occurs frequently with oral and intravenous ribavirin, and most infants treated with the aerosol have not been evaluated 1 to 2 weeks post treatment when anemia is likely to occur. Reticulocytosis has been reported with aerosol use.

Rash and conjunctivitis have been associated with the use of ribavirin aerosol.

Overdosage:

No overdosage with ribavirin by aerosol administration has been reported in the human. The LD_{50} in mice is 2 gm orally. Hypoactivity and gastrointestinal symptoms occurred. In man, ribavirin is sequestered in red blood cells for weeks after dosing.

DOSAGE AND ADMINISTRATION

Before use, read thoroughly the Viratek Small Particle Aerosol Generator (SPAG) Model SPAG-2 Operator's Manual for small particle aerosol generator operating instructions.

Treatment was effective when instituted within the first 3 days of respiratory syncytial virus lower respiratory tract infection.[3] Treatment early in the course of severe lower respiratory tract infection may be necessary to achieve efficacy.

Treatment is carried out for 12-18 hours per day for at least 3 and no more than 7 days, and is part of a total treatment program. The aerosol is delivered to an infant oxygen hood from the SPAG-2 aerosol generator. Administration by face mask or oxygen tent may be necessary if a hood cannot be employed (see SPAG-2 manual). However, the volume of distribution and condensation area are larger in a tent and efficacy of this method of administering the drug has been evaluated in only a small number of patients. Ribavirin aerosol is not to be administered with any other aerosol generating device or together with other aerosolized medications. Ribavirin aerosol should not be used for patients requiring simultaneous assisted ventilation (see Boxed Warnings).

Virazole is supplied as 6 grams of lyophilized drug per 100 ml vial for aerosol administration only. By sterile technique, solubilize drug with sterile USP water for injection or inhalation in the 100 ml vial. Transfer to the clean, sterilized 500 ml widemouth Erlenmeyer flask (SPAG-2 Reservoir) and further dilute to a final volume of 300 ml with sterile USP water for injection or inhalation. The final concentration should be 20 mg/ml. **Important:** This water should not have had any antimicrobial agent or other substance added. The solution should be inspected visually for particulate matter and discoloration prior to administration. Solutions that have been placed in the SPAG-2 unit should be discarded at least every 24 hours and when the liquid level is low before adding newly reconstituted solution.

Using the recommended drug concentration of 20 mg/ml ribavirin as the starting solution in the drug reservoir of the SPAG unit, the average aerosol concentration for a 12 hour period would be 190 micrograms/liter (0.19 mg/l) of air.

HOW SUPPLIED:

Virazole® (ribavirin) Aerosol is supplied in 100 ml glass vials with 6 grams of sterile, lyophilized drug which is to be reconstituted with 300 ml sterile water for injection or sterile water for inhalation (no preservatives added) and administered only by a small particle aerosol generator (SPAG-2). Vials containing the lyophilized drug powder should be stored in a dry place at 15-25°C (59-78°F). Reconstituted solutions may be stored, under sterile conditions, at room temperature (20-30°C, 68-86°F) for 24 hours. Solutions which have been placed in the SPAG-2 unit should be discarded at least every 24 hours.

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KEY TO ABBREVIATIONS, ACRONYMS, & SYMBOLS

a–A = arterial-alveolar (gradient or ratio)
AARC = American Association for Respiratory Care (formerly AART)
AART = American Association for Respiratory Therapy (now AARC)
ABG = arterial blood gas
ACCP = American College of Chest Physicians
AHA = American Hospital Association
AI = artificial intelligence
AIDS = acquired immunodeficiency syndrome
ALS = amyotrophic lateral sclerosis
AMP = Adenosine monophosphate
APRV = airway pressure release ventilation
ARDS = adult respiratory distress syndrome
ARF = acute respiratory failure
ASRA = also see related article
ASRL = also see related letter
ATS = American Thoracic Society
auto-PEEP = unintended positive end-expiratory pressure
B-P = bronchopleural (eg, B-P fistula or air leak)
BPD = bronchopulmonary dysplasia
CAI = computer-assisted instruction
CCC = chondroplasia calcificans congenita
CDC = Centers for Disease Control
CINAHL = Cumulative Index to Nursing & Allied Health Literature
CLD = chronic lung disease
CO = carbon monoxide
COLD = chronic obstructive lung disease
COP = colloid oncotic pressure
COPD = chronic obstructive pulmonary disease
CO_2 = carbon dioxide
CPAP = continuous positive airway pressure
CPR = cardiopulmonary resuscitation
CPT = chest physical therapy
CT = computed x-ray tomography
$D_{LCO\text{-}sb}$ = single-breath diffusion of carbon monoxide across the lung
DME = durable medical equipment
DRG = diagnosis-related group
ECMO = extracorporeal membrane oxygenation
EIB = exercise-induced bronchospasm
EOA = esophageal obturator airway
EPAP = end-positive airway pressure
FDA = Food & Drug Administration
$FEF_{25\text{-}75\%}$ = mid forced expiratory flow
FEV = forced expiratory volume
FEV_1 = forced expiratory volume in 1 second
F_{IO_2} = fraction of inspired oxygen
FRC = functional residual capacity
FVC = forced vital capacity
HCFA = Health Care Financing Administration
HFALV = high-frequency alternating lung ventilation
HFV = high-frequency ventilation
HFJV = high-frequency jet ventilation
HFO = high-frequency oscillation
HFPPV = high-frequency positive-pressure ventilation
HFPV = high-frequency percussive ventilation
HIV = human immunodeficiency virus
HMD = hyaline membrane disease
HME = heat & moisture exchanger (artificial nose)
HMEF = heat & moisture exchanging filter
ICP = intracranial pressure
ICU = intensive care unit
I-E = inspiration-expiration (ratio)
ILD = interstitial lung disease
IMV = intermittent mandatory ventilation
IPPB = intermittent positive-pressure breathing
MIGET = multiple inert gas elimination technique
MIP = maximal inspiratory pressure
MLT = minimal leak technique (of cuff inflation)
MRI = magnetic resonance imaging
MV = mechanical ventilation
NBRC = National Board for Respiratory Care (formerly NBRT)
NBRT = National Board for Respiratory Therapy (now NBRC)
NFPA = National Fire Protection Association
NG = nasogastric (tube)
NHLBI = National Heart Lung & Blood Institute
NIH = National Institutes of Health
NOTT = Nocturnal Oxygen Therapy Trial
NO_2 = nitrous oxide
OSA = obstructive sleep apnea
O_2 = oxygen
$P_{(A-a)O_2}$ = alveolar-arterial oxygen-tension difference
P_{aCO_2} = arterial carbon dioxide tension
P_{aO_2} = arterial oxygen tension
PCP = *Pneumocystis carinii* pneumonia
PDA = patent ductus arteriosus
PEEP = positive end-expiratory pressure
PFC = persistent fetal circulation
PFT = pulmonary function test or testing
PIE = pulmonary interstitial emphysema
P_{O_2} = oxygen tension
PSV = pressure-support ventilation
$P_{\bar{v}O_2}$ = mixed venous oxygen tension
RC = respiratory care
RCP = respiratory care practitioners
RDS = respiratory distress syndrome (of infants)
RICU = respiratory intensive care unit
RIP = respiratory inductive plethysmography
RQ = respiratory quotient
S_{aO_2} = arterial oxygen saturation
SCCM = Society for Critical Care Medicine
SI = Système International d'Unités (a system of units of measure)
SIDS = sudden infant death syndrome
SIMV = synchronized intermittent mandatory ventilation
S_{pO_2} = saturation measured via pulse oximetry
$S_{\bar{v}O_2}$ = mixed venous oxygen saturation
tcP_{O_2} = transcutaneous oxygen tension
T-E = tracheoesophageal (fistula)
TGV = thoracic gas volume
TLC = total lung capacity
TYRS = Test Your Radiologic Skill (a Journal feature)
USP = United States Pharmacopeia
VA = Veterans Administration
VC = vital capacity
V_{CO_2} = carbon dioxide production
VDR = volumetric diffusion respiration
V/Q = ventilation-perfusion ratio
V_T = tidal volume
WOB = work of breathing

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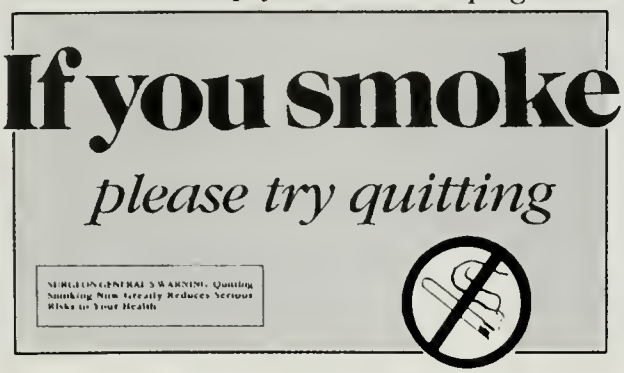

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Risk of Infection During CPR Training and Rescue: Supplemental Guidelines

The Emergency Cardiac Care Committee of the American Heart Association

The American Heart Association and the American Red Cross used the findings from the 1985 National Conference on Standards and Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care to establish their policy on the risk of infection during cardiopulmonary resuscitation (CPR) training and rescue. Findings that support the safety of CPR training and rescue and appropriate risk reduction strategies are presented as an update to the 1985 article. The Emergency Cardiac Care Committee of the American Heart Association incorporated recent advisories from the Centers for Disease Control as well as other information into guidelines that augment earlier recommendations.

(*JAMA*. 1989;262:2714-2715)

FINDINGS from the 1985 National Conference on Standards and Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care are the basis of the official position of both the American Heart Association and the American Red Cross on risk of infections during cardiopulmonary resuscitation (CPR) training and rescue. These guidelines[1] have been augmented by advisories from the Centers for Disease Control released in 1987[2] and 1988[3] and from the National Institute of Occupational Safety and Health in 1989.[4]

For editorial comment see p 2732.

The Emergency Cardiac Care Committee of the American Heart Association considered specific issues related to risk of infection associated with both training and on-site rescue. The American Heart Association and the American Red Cross recommend the following guidelines for adoption.

GUIDELINES FOR RESCUERS WITH KNOWN OR SUSPECTED INFECTIONS

Transmission of hepatitis B virus (HBV) between health care workers and patients has been documented. Instruments and patients' open wounds have been contaminated when health care workers with high concentrations of HBV (much higher than that achieved in human immunodeficiency virus [HIV] infections) in their blood sustained a puncture wound while performing invasive procedures or had weeping lesions or small lacerations on their hands. Transmission of HIV from patients to health care workers has been documented in cases of blood exchange or penetration of the skin by blood-contaminated instruments.[5]

Direct mouth-to-mouth resuscitation will likely result in exchange of saliva between victim and rescuer. Hepatitis B–positive saliva has not been shown to be infectious, however, when applied to oral mucous membranes or through contamination of shared musical instruments or CPR training mannequins used by hepatitis B carriers. In addition, saliva has not been implicated in the transmission of HIV after bites, percutaneous inoculation, or contamination of cuts and open wounds with saliva from HIV-infected patients.[6,7]

Performance of mouth-to-mouth resuscitation or invasive procedures can result in exchange of blood between victim and rescuer if either has had breaks in the skin on or around the lips or soft tissues of the oral cavity mucosa. Thus, there is a theoretical risk of HBV and HIV transmission during mouth-to-mouth resuscitation.[8] It is important to note that the theoretical risk of infection is greater for salivary or aerosol transmission of herpes simplex and *Neisseria meningitidis* and for transmission of airborne diseases such as tuberculosis and respiratory infections.

- *Regardless of the type of infection, rescuers who have an infection that may be transmitted by blood or saliva or who believe they have been exposed to such an infection should not perform mouth-to-mouth resuscitation if circumstances allow other immediate or effective methods of ventilation, such as use of a bag-valve mask.*

GUIDELINES FOR RESCUERS WITH A DUTY TO PROVIDE CPR

The probability of a rescuer's becoming infected with HBV or HIV as a result of performing CPR is minimal.[4] To date, transmission of HBV or HIV infection during mouth-to-mouth resuscitation has not been documented.[9] However, to minimize the risk of transmitting a variety of diseases, mechanical ventilation or barrier devices should be accessible to those asked to provide CPR in the course of their employment. This includes emergency medical service personnel, firefighters, police, and lifeguards, as well as hospital and clinic health care workers.

Although efficacy in preventing disease transmission has not been demonstrated conclusively, masks with one-way valves and bag-valve devices are available, and those with a duty to respond should be instructed in their use during training. Plastic mouth and nose covers with filtered openings also are available and *may* provide a degree of protection against transfer of oral fluids and aerosols. Masks without one-way valves (including those with S-shaped mouthpieces) and handkerchiefs offer little, if any, protection and should not be considered for routine use. Intubation obviates the need for mouth-to-mouth resuscitation and is more effective than the use of bag-mask devices. Early intubation should be encouraged when equipment and trained professionals are available.

- *Individuals with a duty to respond are reminded of their moral, ethical, and, in certain situations, legal obligations to provide CPR, especially in the occupational setting.*

GUIDELINES FOR THE LAYPERSON

The layperson who responds in an emergency should be guided by individ-

From the Emergency Cardiac Care Committee, American Heart Association, Dallas, Tex

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ual moral and ethical values and knowledge of risks that may exist in various rescue situations. It is safest for the rescuer to assume that all emergency situations that involve transfer of certain body fluids have the potential for disease transmission for both rescuer and victim.

Intact skin is the primary defense against transmission of blood-borne diseases during CPR. Transmission of HBV or HIV is more likely if the rescuer has lesions, cuts, or sores in or around the mouth or on the hands and has contact with the victim's blood, vomitus that contains blood, and/or saliva that contains blood. A rescuer who believes he or she has had parenteral or mucous membrane contact with the victim's blood or blood-contaminated body fluids should wash promptly and thoroughly and contact a physician.

• *As a minimum action, in situations perceived as high-risk for disease transmission, the lay rescuer should assess the victim's responsiveness, call for help, position the victim, open the airway, and, in the absence of a pulse, perform chest compressions.*[10] *However, the rescuer should remember that delayed ventilation could mean death or disablement for an otherwise healthy person, while risk to the rescuer, even with a known HBV/HIV-positive victim, is considered very low.*

CPR TRAINING FOR INFECTED INDIVIDUALS

To date, transmission of HBV infection through use of CPR mannequins by HBV carriers has not been shown. Neither has saliva been implicated in HIV transmission. Because of potential breakdown of oral and circumoral mucosa during practice on a mannequin, however, CPR training poses a theoretical risk to class participants. It is recommended that students and instructors adhere to the guidelines that follow.

Acute Infections or Dermatologic Lesions

Acute respiratory infections such as the common cold run a short course, and most breaks in the skin heal naturally or after medical attention. Therefore, students or instructors should postpone CPR training if they (1) are known to be in the active stage of an infection, (2) have reason to believe they have been exposed to an infectious disease, or (3) have dermatologic lesions on their hands or in oral or circumoral areas.

Chronic Infections

Chronic infections such as HBV and HIV persist over an extended period and can be transmitted even when the carrier is asymptomatic. If an instructor wishes to train or to present course completion cards to an individual with a known chronic infection or if the instructor has a known chronic infection, precautions must be taken to protect other participants from exposure. This is best accomplished by providing the infected individual with a separate mannequin that is not used by anyone else until it has been cleaned according to recommended end-of-class decontamination procedures.

• *An individual who has an acute or chronic infection that may be transmitted by blood or saliva must not participate in CPR training until his or her personal physician has reviewed the circumstances carefully and indicated whether participation is appropriate. Because CPR course participants may not know that they have been exposed to an infection, it is imperative that participants and instructors adhere strictly to established procedures for decontamination of the mannequin.*[11] *In addition, requests for individual mannequins should be honored, within reason. Equitable accommodations for all participants in CPR programs are encouraged.*

EDUCATION FOR THE CHRONICALLY INFECTED RESCUER

Individuals with chronic infections should be educated about potential transmission of infection to victims during CPR. Course participants should be made aware of guidelines for rescuers with known or suspected infections. Health risks to the chronically infected rescuer also should be emphasized. If the rescuer's immune system has been altered by any cause, performance of CPR may pose a greater risk to the rescuer. Contact with a victim who is in the active stage of an infection (eg, influenza, tuberculosis, HBV infection, and other respiratory infections) may jeopardize the health of the rescuer with depressed immune function.

GUIDELINES FOR INDIVIDUALS UNABLE TO COMPLETE A CPR COURSE

It is the position of the American Heart Association and the American Red Cross that all reasonable accommodations should be made to provide CPR training to anyone who desires it. It is understood, however, that not everyone will be able to meet the standards required for completion of a CPR course. Such individuals include, but are not limited to, those with physical disabilities that prevent acute ventilation of a mannequin or patient, those unable to perform adequate chest compressions, and those with chronic infections. This may create a dilemma for an individual whose job requires CPR course completion.

Whether an infected worker can care for patients adequately and safely must be determined on an individual basis. The worker's personal physician should make this decision in conjunction with the employing agency and its medical advisers.

• *It is not the role of the American Heart Association or the American Red Cross to lower their course completion standards to accommodate, for purposes of employment, individuals unable to meet these standards. This is an issue that must be resolved by the employer and the employee; thus, the employer must decide whether to waive the CPR course completion requirement. The more important issue for someone who is unable to complete the desired course is whether he or she is able to work in a situation that requires administration of CPR.*

Approved by the Steering Committee of the American Heart Association.

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Calendar of Events

AARC & AFFILIATES

January 18-19, 1990, in Augusta, Georgia. The Georgia Society for Respiratory Care presents its annual Winter Meeting and Awards Banquet. Contact Bob Burnaugh at (404) 944-5189.

January 24-26 in Eugene, Oregon. The Oregon Society for Respiratory Care holds the 1990 Pacific Northwest Respiratory Care Conference at the Eugene Hilton. Call (503) 686-6891 for information.

January 26 in Ruidoso, New Mexico. The NMSRC presents "Winterfest '90" at the Swiss Chalet Inn. Topics focus on current trends in cardiopulmonary care. Contact Robert Quintela or Melanie Brule at (505) 881-9111 or the NMSRC, PO Box 35417, Station D, Albuquerque NM 87176-5417.

February 7-8, 1990, in Salt Lake City, Utah. The Utah Society for Respiratory Care presents its Annual Meeting, "The Future Is Now in Respiratory Care," at the Airport Hilton. Keynote speakers are Irwin Ziment MD, Patrick Dunne MEd RRT, and D Litchford MD. Contact Linda Jordan, Respiratory Care Department, University of Utah Health Science Center, 50 North Medical Dr, Salt Lake City UT 84132. (801) 566-4693.

April 17-19 in Philadelphia, Pennsylvania. The Pennsylvania Society for Respiratory Care presents its Annual Conference and Exhibition at the Adam's Mark Hotel. This year's theme is "Putting on the Ritz for Our 25th!" Respiratory care and cardiovascular technology are highlighted. Contact Jill Bucholtz at (215) 526-3341 or Wendy Janor at (215) 456-8049.

OTHER MEETINGS

March 6-9 in Waterville, New Hampshire. The Respiratory Care Department of Mary Hitchcock Memorial Hospital presents a respiratory care symposium, "1990: Challenges in Critical Care," at the Waterville Valley Conference Center. Contact Office of Continuing Education, Dartmouth-Hitchcock Medical Center, Hanover NH 03756. (603) 646-5744.

August 30-September 8, 1990, Hawaiian cruise aboard the SS Constitution. Dream Cruises' 4th Annual Cruise for Continuing Education presents "Each One, Teach One." Fly to Oahu for two days in Waikiki before boarding ship to four other ports and the islands of Hawaii, Maui, and Kauai. Prices start at $1,215 plus air from your gateway city. Write Dream Cruises, 10882 La Dona Ave, Garden Grove CA 92640. 800-462-3628. CA residents call (714) 636-2566.

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AARC SUMMER FORUM

The Registry Resort, Naples, Florida, July 13-15, 1990
The Westin, Vail, Colorado, July 11-13, 1991

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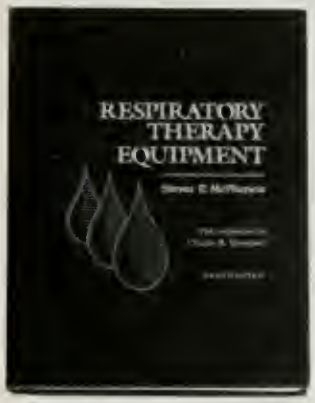

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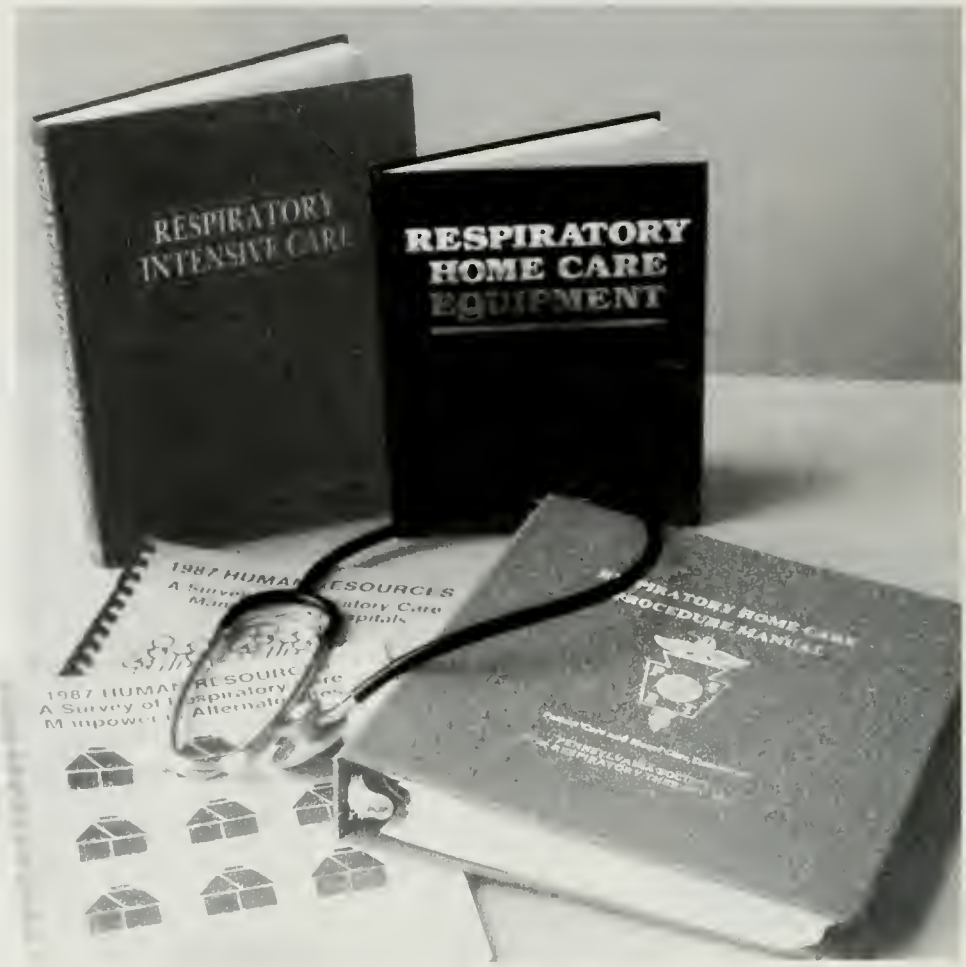

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The abstract will be the only evidence by which the reviewers will decide whether the author should be invited to present a paper at the Open Forum. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An **original study** abstract ***must*** include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

A **method/device evaluation** abstract ***must*** include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience ***or*** a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

A **case report** abstract ***must*** report a case that is uncommon or of exceptional teaching/learning value and must include: (1) case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

An optical scanner will be used to process abstracts. ***Do not use a dot-matrix printer.*** First line of abstract should be the title. Title should explain content. Type the abstract ***double-spaced*** on ***plain white bond paper***, on ***one page*** only (copier bond is excellent). Do not underline or boldface and insert only one letter space between sentences. Provide a 1-inch margin top and bottom, a 1/2 inch left margin, and an approximate 1/2 inch ragged right margin.

No identification of authors or institutions is to appear on the abstract sheet or in the abstract itself. Make the abstract all one paragraph. Data may be submitted in table form ***provided the table width is limited to 60 letter spaces*** (ie, letters or numbers plus necessary blank spaces = 60). ***No*** figures or illustrations are to be attached to the abstract.

Type all information required to complete the author information form on the other side of this page. A photocopy of good quality may be used.

Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained.

Check the abstract for (1) errors in spelling, grammar, facts and figures; (2) clarity of language; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed.

Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

The mandatory Final Deadline is June 9 (postmark). Authors will be notified of acceptance or rejection ***by letter only***—to be mailed by August 15.

Authors may choose to submit abstracts early. Abstracts received by March 26 will be reviewed and the authors notified by May 1. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (June 9).

Mailing Instructions

Mail 1 copy of the abstract, 1 author information sheet, and a stamped, self-addressed postcard (for notice of receipt) to:

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RESPIRATORY CARE

Instructions for Authors and Typists

These Instructions are meant to guide authors and typists, including veterans in those roles, in the production of quality manuscripts. Perfection is not expected, but the well-prepared manuscript has the best chance for prompt review and early publication.

General Requirements

Submissions should (1) be related to respiratory care, (2) be planned for one of the publication categories below, and (3) be prepared as indicated in these Instructions. A letter accompanying the manuscript must specify the intended publication category, be signed by all the authors, and, when there are two or more authors, state that "We, the undersigned, have all participated in the work reported, read the accompanying manuscript, and approved its submission for publication."

Publication Categories

Research Article (Study): A report of an original investigation.
Evaluation of a Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.
Case Report: A report of a clinical case that is uncommon or of exceptional teaching value. The author(s) must have been associated with the case. A case-managing physician must be one of the authors or, if not an author, must supply a letter approving the manuscript.
Case Series: Like a Case Report but including a number of cases.
Review Article: A comprehensive, critical review of the literature and state of the art of a pertinent topic that has been the subject of 40 or more published research papers.
Overview: A critical review of a pertinent topic about which not enough research has been published to merit a Review Article.
Update: A report of subsequent developments in a topic that has been critically reviewed (not necessarily in this journal).
Point of View: A paper expressing the author's personal opinions on a pertinent topic.
Special Article: If a paper does not fit one of the foregoing categories but is pertinent, the editors may consider it as a Special Article.
Editorial: A paper that draws attention to a pertinent concern.
Letter: A signed communication about material published in this journal or on topics of interest or value to readers.
Blood Gas Corner: A brief, instructive case report (real or fictional) involving invasively or noninvasively obtained respiratory care blood data, followed by questions for readers— with answers and discussion.
PFT Corner: Like Blood Gas Corner but involving pulmonary function testing.
Test Your Radiologic Skill: Like Blood Gas Corner and PFT Corner but involving pulmonary-medicine radiography and including one or two 4 × 5 or 5 × 7 inch prints of radiographs. The case must be real.
Review of Book, Film, Tape, or Software: Anyone interested in writing a review can discuss it with an editor.

Editorial Consultation and Author's & Typist's Kit

To discuss a writing project, write to RESPIRATORY CARE, PO Box 29686, Dallas TX 75229 or call 214/243-2272.

Authors are urged to obtain the RESPIRATORY CARE Author's & Typist's Kit. The Kit provides *authors* with specific guidance about writing a research paper, writing a case report, converting to and from SI units, and in-house manuscript review. *Typists* can use the Kit's Model Manuscript, a list of journal name abbreviations, and a copy of these Instructions. The Kit is free from the Journal office.

Preparing the Manuscript

General Concerns ***Typist***

- Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
- Number pages in upper right corner and leave margins of 1½" or more on all four sides of the page.
- For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
- Meticulously follow instructions for typing references.

General Concerns ***Author:***

- Structure manuscript as specified hereafter.
- Provide all requested information on title page as specified hereafter.
- Proofread manuscript for completeness, clarity, grammar, spelling, be sure all references, figures, and tables are cited in the text.
- Consider having paper reviewed in-house before submission.
- Have all co-authors proofread and approve manuscript and sign submission letter.

Manuscript Structure

Most kinds of papers have standard parts in a standard order. However, papers can vary individually, and not every paper will have all the parts listed here.

Research Article: Title page, abstract page, continuous text (Introduction, Materials & Methods, Results, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends. Please consult "Writing a Research Paper," Respir Care 1985;30:1057 (Dec 1985) and Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
Evaluation of Device/Method/Technique: Title page, abstract page, continuous text (Introduction, Description of Device/Method/Technique, Methods of Evaluation, Results of Evaluation, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends.
Case Report or Case Series: Title page, abstract page, continuous text (Introduction, Case Summary, Discussion), Acknowledgments page, references, tables, figure legends. Also see "How To Write a Better Case Report," Respir Care 1982;27:29 (Jan 1982).
Review Article: Title page, Table of Contents page, continuous text (Introduction, History, Review of Literature, State of the Art, Discussion, Summary), references. May include figures & tables. No abstract. Table of Contents optional. Other formats may be appropriate.
Overview, Update, Point of View, or Special Article: Title page, text (introduction, message), references, tables, figure legends. No abstract.
Letter: Title page (provide a title), text, writer's name & affiliation, references. Tables & figures may be included. Double-space everything. Write "For Publication" on title page.

Structure: Important Details

Title Page: List title of paper, all authors' full names, degrees, credential letters, professional positions, and affiliations. List correspondence address, telephone number, and reprint address if desired. Name sources of grants or other support. Identify any author's consulting or commercial relationships that pertain to the paper's topic.

Abstract Page: Number this Page 1. List paper's title but omit authors names. Abstract should be 200 words or less and must be informative, briefly specifying main points of paper, such as methods, results, and conclusions drawn.

Statistical Analysis: In research articles, identify statistical tests and chosen level of significance in the Methods section. In Results section, report actual P values.

Figures (illustrations): All photographs, diagrams, & graphs must be numbered as Figure 1, Figure 2, etc, according to the order in which each is first mentioned in the text. Photographs must be glossy prints 5 × 7 to 8 × 10 inches and should be black & white unless color is essential. Letters and numerals must be neat and large enough to remain legible if figure is reduced in size for publication. Final figures must be of professional quality, but 'rough' sketches may accompany the submitted manuscript, with final figures to be prepared after review. Identify each figure on back with a stick-on label showing figure number and arrow indicating top; omit author's name. Cover label with clear tape so ink will not smudge other prints. Supply three sets of unmounted figures. If figure has been published before, include copyright-holder's written permission to use it.

Figure Legends: List figure legends on a separate page, not on figures. If a figure has been published before, list the source in the legend.

Tables: Type each table on a separate page. Avoid more than 8 columns across. Continue a deep table on following pages. Give each table a number and descriptive title, placed above the table. Double-space ALL lines in tables, including column headings and footnotes.

Drugs: Brand names may be given, but always also show generic names.

Units of Measurement: In addition to conventional units of measure, show SI values and units in brackets after conventional expressions: ie, "PEEP, 10 cm H_2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33:861-873 (Oct 1988).

Commercial Products: If three or fewer commercial products are named in the text, list the manufacturer's name and location in parentheses the first time each is mentioned. If four or more products are named, do not list manufacturers in the text; instead, name the products and manufacturers in a Products Sources list at the end of the text. Provide model numbers when available.

Abbreviations: Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Do not create new abbreviations unless absolutely necessary.

References:

- Use references to support statements of fact, indicate sources of information, or guide readers to further pertinent literature.
- Cite only published works—or works accepted for publication. When listing an accepted but still unpublished work, designate the accepting journal's name, followed by "(in press)."
- In the text, cite references by superscript numerals (half space above text), not in parentheses. The first reference cited in the text is number 1, the next is number 2, etc.
- In the reference list, place the cited works in numerical order.
- For the reference list, obtain author names, article and book titles, dates, volume and page numbers from the *original* cited articles and books, not from secondary sources such as other articles' reference lists, which often are inaccurate.
- Type references in medical-journal style. Examples appear at the end of these Instructions. Abbreviate journal names as in *Index Medicus*. A list of many journal-name abbreviations was published in Respir Care 1988;33:1050 (Nov 1988).
- DOUBLE-SPACE the lines of references.
- List ALL authors' names. Do not use "et al" to substitute for names.
- Identify abstracts, editorials, and letters as such. See examples.

Personal Communications, Unpublished Papers, and Unpublished Observations: List unpublished items in parentheses in the text, not in the reference list.

Examples of How To Type References

Notes: Although the examples here are printed with single-spaced lines, please double-space references in manuscripts. Also, note that words in article and book titles are not capitalized—except proper names.

Standard Journal Article:

1. Shepherd KE, Johnson DC. Bronchodilator testing: An analysis of paradoxical responses. Respir Care 1988;33:667-671.

Corporate Author Journal Article:

2. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in Journal Supplement:

(Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

3. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, suppl):139s-143s.

Abstract in Journal:

(Abstracts are not strong references; when possible, full papers should be cited. When cited, abstracts should be identified as such.)

4. Lippard DL, Myers TF, Kahn SE. Accuracy of pulse oximetry in severely hypoxic infants (abstract). Respir Care 1988;33:886.

Editorial in Journal:

5. Rochester DF. Does respiratory muscle rest relieve fatigue or incipent fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

Letter in Journal:

6. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Personal Author Book:

7. Nunn JF. Applied respiratory physiology. New York: Appleton-Century-Crofts, 1969.

Note: To specify pages cited in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1963:85-95 (series of contiguous pages), 1963:85,95 (separated pages).

Corporate Author Book:

8. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with Editor, Compiler, or Chairman as 'Author':

9. Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in Book:

10. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977:171-223.

Submitting the Manuscript

After preparing the manuscript according to these Instructions, perform a final proofreading and check for accuracy and completeness. Then mail three copies of the manuscript and three sets of figures to RESPIRATORY CARE, PO Box 29686, Dallas TX 75229 (or Federal Express to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229). Manuscript copy on IBM-compatible or Macintosh disks in addition to the requisite three hard copies will facilitate processing (Macintosh preferred). Enclose a letter as specified under **General Requirements** at the beginning of these Instructions. Do not submit material that has been published or is being considered elsewhere.

Author's Checklist

1. Is paper for a listed publication category?
2. Does cover letter meet specifications?
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4. Are all pages double-spaced and numbered?
5. Are all references, figures, and tables cited in the text?
6. Are references typed in requested style?
7. Have SI values been provided?
8. Has all arithmetic been checked?
9. Has manuscript been proofread by all authors?

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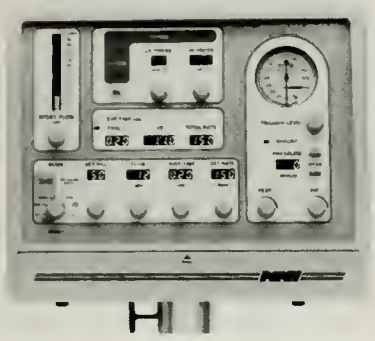

NEONATAL/PEDIATRIC VENTILATOR. The Newport Breeze Model E150 is a microprocessor-controlled, pneumatically powered ventilator intended for neonatal and pediatric patients. According to the manufacturer, the E150's unique design and Intermittent Spontaneous Flow (ISF) system allow true pressure plateau or volume ventilation. MAP (proximal) is displayed; peak or baseline pressures can also be monitored. A self-charging, internal battery provides 1 hour of emergency power. An integral air/O_2 mixer, a comprehensive alarm system, digital displays of key respiratory variables, PEEP/CPAP, adjustable apnea time, adjustable alarm loudness, and reverse I-E ratio ventilation are also standard. Newport Medical Instruments Inc, Dept RC, 300 N Newport Blvd, Newport Beach CA 92663. (800) 451-3111.

AEROSOL INHALATION MONITOR. The AIM (Aerosol Inhalation Monitor) is designed to train patients to use a metered dose inhaler (MDI). According to the manufacturer, the AIM monitors the three main factors needed to use an MDI correctly: inspiratory flow, firing time, and breath-holding time. Red and green lights are used for training, and an accessory that uses cartoon characters makes learning fun. Vitalograph Inc Medical Instrumentation, Dept RC, 8347 Quivira Rd, Lenexa KS 66215. (800) 255-6626, (913) 888-4259.

NASAL CPAP SYSTEM. According to the manufacturer, the Vitalflow nasal CPAP system incorporates several features that facilitate patient comfort and compliance: independent control of flow and pressure that minimizes upper-airway dryness and operation noise; soft nasal masks (small, medium, and large—each available with two facial-fit contours) designed to provide a consistent, uniform and leak-free seal; and a small, lightweight, adjustable CPAP valve capable of providing up to 18-cm H_2O. Two models of the Vitalflow are available: Vitalflow I (for home use) and Vitalflow III (for clinical use). Vitalflow III is equipped with a pressure transducer (with digital readout) for monitoring CPAP and with a port for supplemental oxygen delivery. Vacumetrics Inc, Vacumed Div, Dept RC, 5770 Nicolle St, Ventura CA 93003. (805) 644-7461.

CPR/ACLS LEARNING SYSTEM. Approved by the American Heart Association (AHA), this learning system (consisting of a microcomputer, videodisc player, random-access audio tape player, and 2 video monitors) enables students to train and test for CPR and ACLS certification at their own pace and on their own time; the instructor, or learning system coordinator, needs only to check the student's test results and sign the certification card. The CPR and Intubation manikins that can be interfaced with the system are equipped with electronic sensors that communicate with the computer. The ACLS series consists of four interactive videodisc courses: Arrhythmia Recognition, Airway Management, Megacode, and Circulatory Adjuncts and Resuscitation Pharmacology. In addition to the 14 hours of didactic material included in the courses, the student has access to all ACLS test stations, drug and algorithm reference banks, quizzes, emergency scenario simulations, sensorized manikin practices, and procedure demonstrations. Actronics Inc, Dept RC, 810 River Ave, Pittsburgh PA 15212. (412) 231-6200.

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I. Type of Instn/Practice
1 ☐ Hosp 500 or more beds
2 ☐ Hosp 300 to 500 beds
3 ☐ Hosp 200 to 300 beds
4 ☐ Hosp 100 to 200 beds
5 ☐ Hosp 100 or less beds
6 ☐ Clinic/Group Practice
7 ☐ Independent RT Provider
8 ☐ Industry (Mfgr/Sales)

II. Department
A ☐ Respiratory Ther
B ☐ Cardiopulmonary
C ☐ Anesthesia Service
D ☐ Emergency Dept

III. Specialty
1 ☐ Clinical Practice
2 ☐ Perinatal Pediatrics
3 ☐ Critical Care
4 ☐ Clinical Research
5 ☐ Pulmonary Func Lab
6 ☐ Home Care/Reheb
7 ☐ Education
8 ☐ Management

IV. Position
A ☐ Dept Head
B ☐ Chief Therapist
C ☐ Supervisor
D ☐ Staff Technician
E ☐ Staff Therapist
F ☐ Educator
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I. Type of Instn/Practice
1 ☐ Hosp 500 or more beds
2 ☐ Hosp 300 to 500 beds
3 ☐ Hosp 200 to 300 beds
4 ☐ Hosp 100 to 200 beds
5 ☐ Hosp 100 or less beds
6 ☐ Clinic/Group Practice
7 ☐ Independent RT Provider
8 ☐ Industry (Mfgr/Sales)

II. Department
A ☐ Respiratory Ther
B ☐ Cardiopulmonary
C ☐ Anesthesia Service
D ☐ Emergency Dept

III. Specialty
1 ☐ Clinical Practice
2 ☐ Perinatal Pediatrics
3 ☐ Critical Care
4 ☐ Clinical Research
5 ☐ Pulmonary Func Lab
6 ☐ Home Care/Reheb
7 ☐ Education
8 ☐ Management

IV. Position
A ☐ Dept Head
B ☐ Chief Therapist
C ☐ Supervisor
D ☐ Staff Technician
E ☐ Staff Therapist
F ☐ Educator
G ☐ Medical Director
H ☐ Anesthesiologist
I ☐ Other MD
J ☐ Nurse

V. Are you a member of the AAR
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22mm Female/15mm Male fitting.

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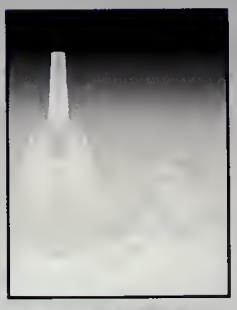
Handy color reference chart and six color-coded resistors make setting level of inspiratory resistance easy and help prevent tampering with prescribed level. The IMT has a capped port for NIF measurement or low-flow oxygen delivery and a 15mm F port for direct connection to the endotracheal airway.



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